

# RHEUMATOLOGY 2024









#### Question 1 of 178





A 65-year-old woman is referred to the rheumatology outpatient department by her general practitioner complaining of bilateral knee pain, worse on mobilization. She has a past medical history of hypertension. She takes amlodipine. She has smoked five cigarettes per day for 20 years and she does not drink alcohol. She works as a cleaner.

On examination, the knees are mildly swollen bilaterally and there is patellofemoral crepitus. There is no joint swelling or tenderness elsewhere. Cardiorespiratory examinations are unremarkable.

What is the most appropriate management?

Codeine phosphate	
Local muscle strengthening exercises	
Methotrexate	
Oral NSAID	
Prednisolone	

Submit answer

Reference ranges V

Score: **0% 1** -







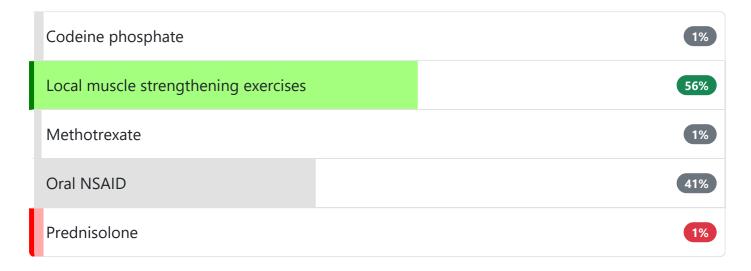
Question 1 of 178



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On examination, the knees are mildly swollen bilaterally and there is patellofemoral crepitus. There is no joint swelling or tenderness elsewhere. Cardiorespiratory examinations are unremarkable.

What is the most appropriate management?



Local muscle strengthening exercises and general aerobic fitness is an important component of knee and hip osteoarthritis

Important for me Less important



Local muscle strengthening exercises and general aerobic fitness is the correct answer. The patient presents with bilateral knee pain with mild swelling, worse on mobilization and there is clinical evidence of crepitus. These symptoms and signs are consistent with a diagnosis of osteoarthritis of the knees. The initial management of osteoarthritis of the knees consists of advice regarding local muscle strengthening exercises and general aerobic fitness +/- paracetamol and topical ibuprofen.

Codeine phosphate is incorrect. This is a potential option if there is ongoing pain despite paracetamol, topical ibuprofen and oral ibuprofen but would not be first-choice.

Methotrexate is incorrect. The pain of rheumatoid arthritis tends to be worse at rest rather than on activity and there is no mention of diurnal variation or early morning stiffness in this case. This is a medication used in maintenance of remission in rheumatoid arthritis. Smoking is a risk factor for this condition.

Prednisolone is incorrect. This patient has osteoarthritis, not rheumatoid arthritis and therefore there is no place for oral steroids.

Oral NSAID is incorrect. This is an option for the management of knee osteoarthritis after failure of exercises, oral paracetamol and topical ibuprofen.

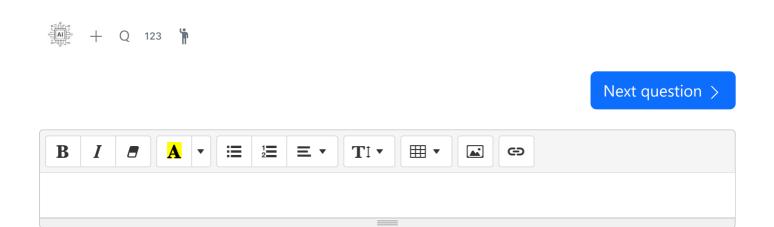


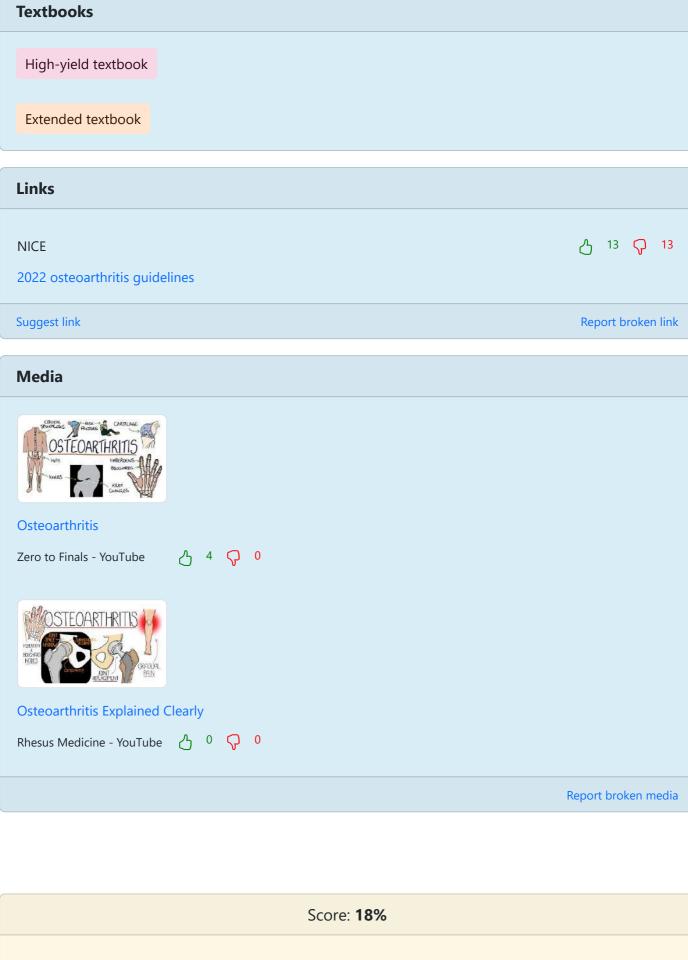
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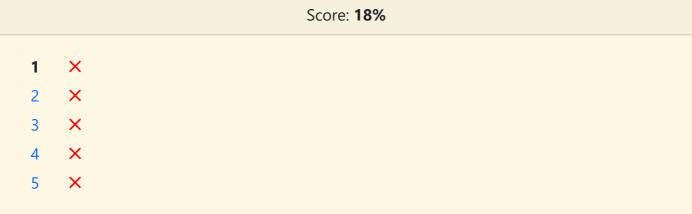
# Osteoarthritis: management \*

NICE updated its guidelines on the management of osteoarthritis (OA) in 2022

- all patients should be offered help with weight loss, given advice about local muscle strengthening exercises and general aerobic fitness
- topical NSAIDs are first-line analgesics. Topical NSAIDs may be particually beneficial for patients with OA of the knee or hand NICE
- second-line treatment is oral NSAIDs
  - o a proton pump inhibitor should be co-prescribed with NSAIDs
  - these drugs should be avoided if the patient takes aspirin
- NICE recommend we do not offer paracetamol or weak opioids, unless: NICE
  - they are only used infrequently for short-term pain relief and
  - o all other pharmacological treatments are contraindicated, not tolerated or ineffective
- glucosamine and strong opioids are not recommended
- non-pharmacological treatment options include walking aids for knee and hip OA
- intra-articular steroid injections may be tried if standard pharmacological treatment is ineffective
  - patients should be aware that they only provide short-term relief (2-10 weeks)
- if conservative methods fail then refer for consideration of joint replacement







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#### Question 2 of 178





A 68-year-old woman presents to a care of the elderly clinic. This is her first attendance following recent frequent falls at home. Her most recent fall was two weeks ago during which time she had a CT head/c-spine/right hip which was negative for any acute injury.

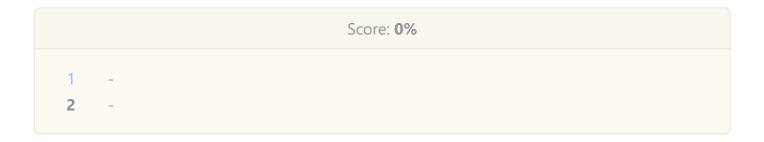
Her only symptom between falls is a pain in her right hip which has been worsening over the past year. This is worse on activity and in the evenings. She is tender to deep palpation and has painful internal and external rotation on examination. Otherwise, she reports that she is well in herself and her only past medical history includes obesity and type 2 diabetes mellitus. She has been advised by her general practitioner to lose weight.

Which of the following is the most appropriate additional advice for her hip pain?

<ul> <li>Muscle strengthening exercises and aerobic fitness</li> <li>Reduce alcohol intake</li> <li>Regular rest throughout the day</li> </ul>	Elevate lower limbs when seated	
	Muscle strengthening exercises and aerobic fitness	
Regular rest throughout the day	Reduce alcohol intake	
	Regular rest throughout the day	
Smoking cessation	Smoking cessation	

Submit answer

Reference ranges ∨









Ouestion 2 of 178



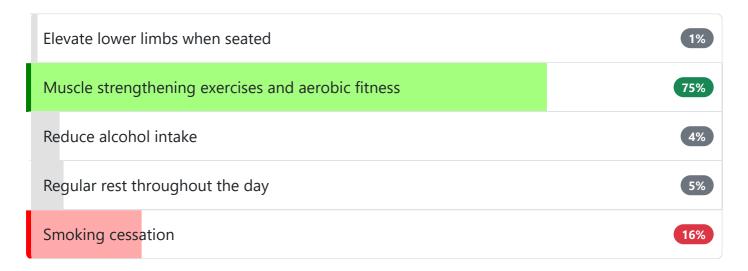
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Which of the following is the most appropriate additional advice for her hip pain?



Local muscle strengthening exercises and general aerobic fitness is an important component of knee and hip osteoarthritis

Important for me Less important



This woman has osteoarthritis of the right hip. This is evidenced by the chronicity and nature of the pain. These factors, as well as her recent negative imaging, make acute injury unlikely. As per the latest NICE guidance, all patients diagnosed with osteoarthritis of the knee or hip should be offered help with weight loss, given advice about local muscle strengthening exercises and general aerobic fitness.

Elevating limbs is helpful in acute injury to reduce acute swelling but will not affect this woman's osteoarthritis.

Reducing alcohol intake is a preventative measure for gout however would not be effective in this case.

Regular rest throughout the day may temporarily prevent symptoms but will be debilitating and not result in any lasting improvement.

Smoking cessation is useful in rheumatoid arthritis however the history here is more classical of osteoarthritis.

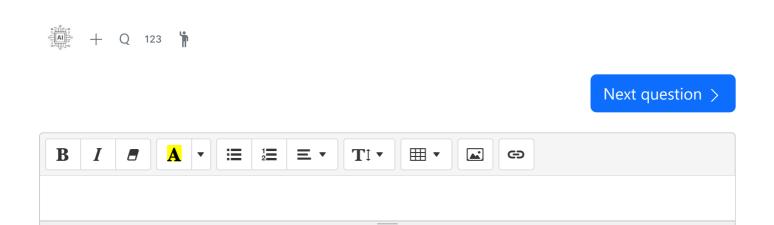


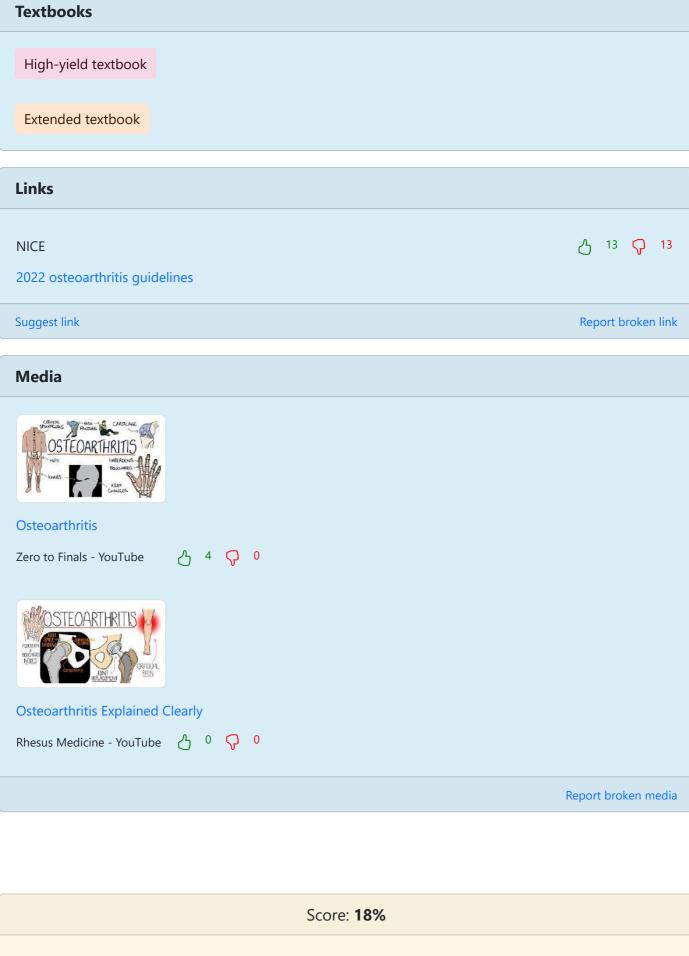
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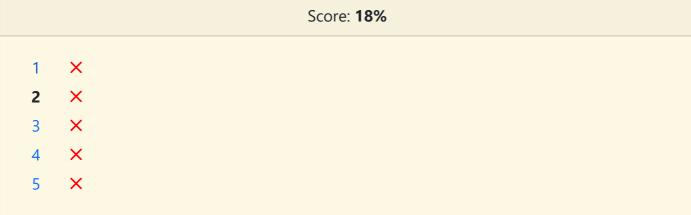
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#### Question 3 of 178





A 28-year-female presents with increasing fatigue. She also complains of joint pains affecting several joints. There is associated joint stiffness lasting about 20-30 mins. She also gets a skin rash, especially in sun-exposed areas. There is no significant past medical history and she is not using any regular medications. She works as an office clerk and the joint pains are making it difficult for her to continue her work normally. Following are the results of her investigations:

Hb	9.3 g/l	Na <sup>+</sup>	136 mmol/l	Bilirubin	98 µmol/l
Platelets	99 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.2 mmol/l	ALP	89 u/l
WBC	7.3 * 10 <sup>9</sup> /l	Urea	4.5 mmol/l	ALT	23 u/l
Neuts	3.4 * 10 <sup>9</sup> /l	Creatinine	87 µmol/l	γGT	43 u/l
Lymphs	2.7 * 10 <sup>9</sup> /l	CRP	<1 mg/l	Albumin	32 g/l
Eosin	0.2 * 10 <sup>9</sup> /l	C3, C4	low		

Chest x-ray and ECG were normal. Urine exam showed protein +++.

Which of the following is a feature of the underlying condition in this patient?

ANA usually negative	
Absence of a relapsing and remitting course	
Lower risk of thrombo-embolism	
Normal CRP	
Anti-CCP antibody level is highly specific	

Submit answer

Reference ranges ∨

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Question 3 of 178



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Hb	9.3 g/l	Na <sup>+</sup>	136 mmol/l	Bilirubin	98 µmol/l
Platelets	99 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.2 mmol/l	ALP	89 u/l
WBC	7.3 * 10 <sup>9</sup> /l	Urea	4.5 mmol/l	ALT	23 u/l
Neuts	3.4 * 10 <sup>9</sup> /l	Creatinine	87 µmol/l	γGT	43 u/l
Lymphs	2.7 * 10 <sup>9</sup> /l	CRP	<1 mg/l	Albumin	32 g/l
Eosin	0.2 * 10 <sup>9</sup> /l	C3, C4	low		

Chest x-ray and ECG were normal. Urine exam showed protein +++.

Which of the following is a feature of the underlying condition in this patient?

ANA usually negative	5%
Absence of a relapsing and remitting course	14%
Lower risk of thrombo-embolism	2%
Normal CRP	60%
Anti-CCP antibody level is highly specific	19%

A raised CRP in a patient with known SLE may indicate an underlying infection

Important for me Less important



Systemic Lupus Erythematosus (SLE) is the most likely diagnosis in this patient as there are several pointers towards it including female sex, joint pains, photosensitive rash, haemolytic anaemia, thrombocytopenia, normal CRP, low complement level, and proteinuria.

ANA is positive in more than 90-95% of patients with SLE. Hence it's highly unlikely to have SLE

with a negative ANA.

SLE is a relapsing and remitting disease.

SLE is associated with an increased risk of thrombosis.

CRP is normal in SLE unless there is a secondary infection.

Anti-CCP has high specificity for rheumatoid arthritis rather than SLE.



Next question >

# Systemic lupus erythematosus: investigations

#### **Antibodies**

- 99% are ANA positive
  - o this high sensitivity makes it a useful rule out test, but it has low specificity
- 20% are rheumatoid factor positive
- anti-dsDNA: highly specific (> 99%), but less sensitive (70%)
- anti-Smith: highly specific (> 99%), sensitivity (30%)
- also: anti-U1 RNP, SS-A (anti-Ro) and SS-B (anti-La)

#### Monitoring

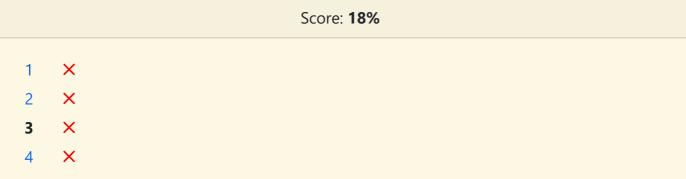
- inflammatory markers
  - ESR is generally used
  - during active disease the CRP may be normal a raised CRP may indicate underlying infection
- complement levels (C3, C4) are low during active disease (formation of complexes leads to consumption of complement)
- anti-dsDNA titres can be used for disease monitoring (but note not present in all patients)



Next question >



# **Textbooks** High-yield textbook Extended textbook Links △ 7 ♀ 5 American College of Rheumatology Systemic Lupus Erythematosus diagnostic criteria Suggest link Report broken link Media Systemic Lupus Erythematosus (SLE) - signs and symptoms, pathophysiology, investigations, treatment 占 1 ♀ 0 Armando Hasudungan - YouTube SYSTEMIC LUPUS ERYTHEMATOSUS Systemic lupus erythematosus (SLE) - causes, symptoms, diagnosis & pathology 少 ○ ♀ ○ Osmosis - YouTube Report broken media Score: 18%



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#### Ouestion 4 of 178





A 28-year-old woman presents to the medical take with a macular rash, and arthralgia, which she reports have been ongoing for the past three weeks. She had initially attributed the symptoms to a viral infection, however, when her symptoms failed to improve after a week and she continued to experience fevers of > 39°C she visited her GP, who referred her to the medical team for further investigations. She denied any early morning stiffness, gastrointestinal upset, dysuria or visual symptoms and prior to the onset of her symptoms was otherwise in good health.

She is a non-smoker, drinks occasional alcohol and has travelled to France and to Spain in the last year. She reports no family history of note and has no risk factors for HIV or tuberculosis aside from previous travel to India 5 years previously. She works as a financial analyst and managed to return to work after 1 week of sick leave, although she has not been able to pursue her usual sporting activities.

On examination, she has a widespread macular rash with minimal signs of excoriation. She has an erythematous oropharynx with no signs of exudate and no significant lymphadenopathy. Cardiovascular, respiratory, abdominal and neurological examinations are unremarkable. Examination of the joints demonstrates no signs of active synovitis and in spite of tenderness in the shoulders and knees, there are no areas of restricted active or passive range of movement.

#### Her blood tests are detailed below:

Hb	130 g/L	Male: (135-180) Female: (115 - 160)
Platelets	550 * 10 <sup>9</sup> /L	(150 - 400)
WBC	15.4 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Neuts	13.1 * 10 <sup>9</sup> /L	(2.0 - 7.0)
Lymphs	1.6 * 10 <sup>9</sup> /L	(1.0 - 3.5)
Mono	0.5 * 10 <sup>9</sup> /L	(0.2 - 0.8)
Eosin	0.2 * 10 <sup>9</sup> /L	(0.0 - 0.4)
Na <sup>+</sup>	140 mmol/L	(135 - 145)
K <sup>+</sup>	4.0 mmol/L	(3.5 - 5.0)
Urea	4.0 mmol/L	(2.0 - 7.0)
Creatinine	70 μmol/L	(55 - 120)
Bilirubin	15 μmol/L	(3 - 17)
ALP	70 u/L	(30 - 100)
ALT	35 u/L	(3 - 40)
ANA	1:20	(< 1:40)

Rheumatoid factor	5 IU/ml	(<14)
CRP	81 mg/L	(< 5)
INR	1.0	(0.9 - 1.3)

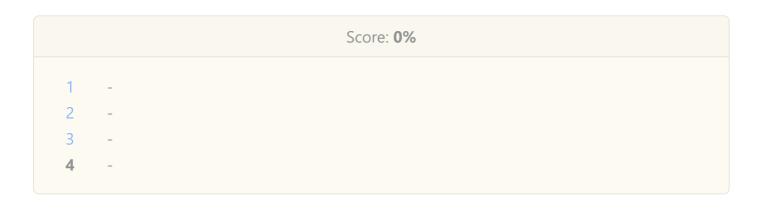
A full autoimmune screen along with Lyme serology, HIV and hepatitis screen are all unremarkable and a CT chest/abdomen/pelvis reveals no significant abnormalities. An echocardiogram shows no signs of infective endocarditis and 3 consecutive blood cultures performed off antibiotics are negative. A blood film confirms neutrophilia.

Given the likely diagnosis, what would be the first-line treatment for this patient?

Anakinra	
Methotrexate	
Methylprednisolone	
Non-steroidal anti-inflammatory drugs (NSAIDs)	
Prednisolone	

### Submit answer

Reference ranges ∨





Question 4 of 178







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Eosin	0.2 * 10 <sup>9</sup> /L	(0.0 - 0.4)		
Na <sup>+</sup>	140 mmol/L	(135 - 145)		
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Creatinine	70 μmol/L	(55 - 120)		
Bilirubin	15 μmol/L	(3 - 17)		
ALP	70 u/L	(30 - 100)		
ALT	35 u/L	(3 - 40)		
ANA	1:20	(< 1:40)		

Rheumatoid factor	5 IU/ml	(<14)	
CRP	81 mg/L	(< 5)	
INR	1.0	(0.9 - 1.3)	

A full autoimmune screen along with Lyme serology, HIV and hepatitis screen are all unremarkable and a CT chest/abdomen/pelvis reveals no significant abnormalities. An echocardiogram shows no signs of infective endocarditis and 3 consecutive blood cultures performed off antibiotics are negative. A blood film confirms neutrophilia.

Given the likely diagnosis, what would be the first-line treatment for this patient?

Anakinra	8%				
Methotrexate	5%				
Methylprednisolone	7%				
Non-steroidal anti-inflammatory drugs (NSAIDs)	56%				
Prednisolone	23%				
NSAIDs are the first line treatment for Still's disease, not steroids					



Important for me Less important

This patient has adult-onset Still's disease as suggested by the Yamaguchi criteria - this patient has four of the major criteria (fever >39 °C, for over 1 week, arthralgia or arthritis for more than two weeks, rash, leukocytosis with >80% polymorphonuclear cells) and one of the minor criteria (negative rheumatoid factor and negative ANA).

Adult-onset Still's disease is effectively a diagnosis of exclusion and malignancy, autoimmune and infection must be ruled out.

This patient's presentation would be described as mild disease as she has no evidence of organ involvement and has been able to continue with her daily life to a large extent. Therefore, NSAIDs should be considered first-line. Should she see no improvement or limited improvement after 10 days - 2 weeks of regular NSAID therapy, oral steroids would be considered.

In patients with moderate disease, i.e. evidence of organ involvement and/or very limited by their symptoms, steroids would be considered first-line.

In patients with severe or life-threatening disease i.e. significant organ dysfunction should be

commenced on IV steroids.

In patients who require ongoing steroids to control their symptoms or in whom steroids do not control symptoms, disease-modifying agents such as anakinra or methotrexate should be considered.



Next question >

#### Still's disease in adults \*

#### Epidemiology

has a bimodal age distribution - 15-25 yrs and 35-46 yrs

#### **Features**

- arthralgia
- elevated serum ferritin
- rash: salmon-pink, maculopapular
- pyrexia
  - o typically rises in the late afternoon/early evening in a daily pattern and accompanies a worsening of joint symptoms and rash
- lymphadenopathy
- rheumatoid factor (RF) and anti-nuclear antibody (ANA) negative

The diagnosis of Still's disease in adults can be challenging. The Yamaguchi criteria is the most widely used criteria and has a sensitivity of 93.5%.

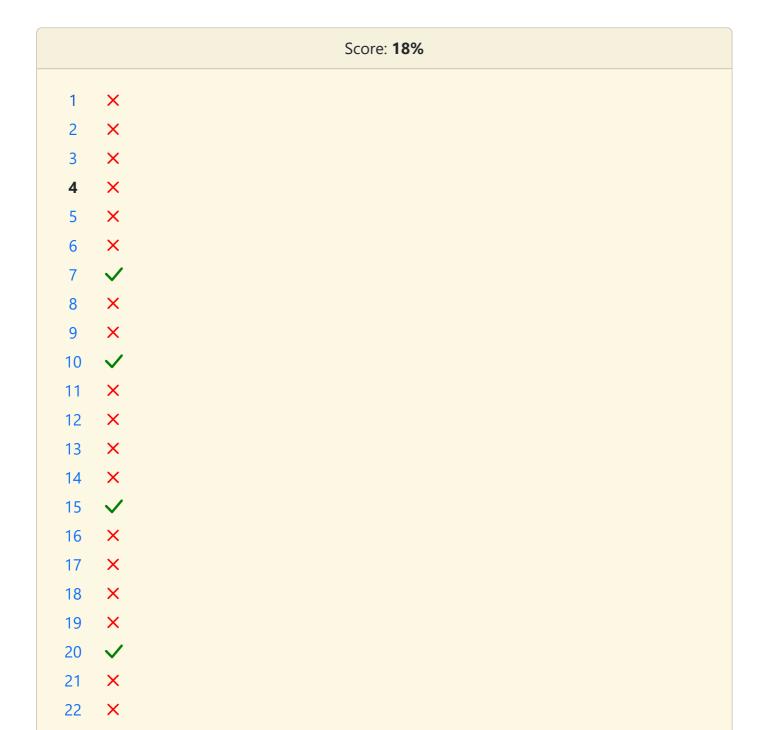
#### Management

- NSAIDs
  - should be used first-line to manage fever, joint pain and serositis
  - o they should be trialled for at least a week before steroids are added.
- steroids
  - o may control symptoms but won't improve prognosis
- if symptoms persist, the use of methotrexate, IL-1 or anti-TNF therapy can be considered









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#### Question 5 of 178

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A 47-year-old lady presented with a three-week history of pain in her fingers. She had noticed her hands were getting extremely cold when she went outside and turned a 'funny colour'. When she came back inside her hands were very painful as they began to warm up. She had managed in the past by wearing gloves outside but now had ulcers on her fingertips which she had never experienced before. She also complained of epigastric pain and had longstanding shortness of breath.

Her past medical history included pulmonary fibrosis and hypertension. Her medications included propranolol, amlodipine, simvastatin and omeprazole.

On examination the skin over her hands was dry and shiny and there was severe digital ulceration on three fingertips of the left hand. There was no exudate or erythema. The fingertips were dusky in colour and extremely tender. The skin over the upper arms and chest appeared normal. On auscultation of the lungs there were fine bibasal inspiratory crepitations which did not alter in character upon coughing. Heart sounds were normal with no added murmurs. There was a left ventricular heave

Which of the following is the most appropriate management plan for this lady?

	Start flucloxacillin and stop all anti-hypertensive medications	
dos	Educate this lady about the use of gloves and hand-warmers and increase her amlodipi	ne ×
	Stop amlodipine and refer for an urgent dermatology assessment	
	Stop propranolol and admit for an iloprost infusion	
	Start high dose oral prednisolone	

Submit answer

Reference ranges  $\vee$ 

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Question 5 of 178

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Which of the following is the most appropriate management plan for this lady?

Start flucloxacillin and stop all anti-hypertensive medications				
Educate this lady about the use of gloves and hand-warmers and increase hamlodipine dose	ner 22%			
Stop amlodipine and refer for an urgent dermatology assessment				
Stop propranolol and admit for an iloprost infusion	56%			
Start high dose oral prednisolone	18%			

Epoprostenol (amongst other prostaglandins) can be used in the treatment of Raynaud's phenomenon

Important for me Less important



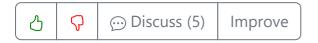
This patient has secondary Raynauds associated with an underlying diagnosis of Limited Systemic Sclerosis. Her disease is 'limited' as opposed to 'diffuse' as she does not have skin changes

proximal to the elbows. She clearly has systemic involvement with pulmonary fibrosis and oesophageal dysmotility (she has epigastric pain and is taking omeprazole). Other features that were not mentioned in the question but may be present in such patients include telangiectasia, typically over mucosal surfaces, a 'beak-like' nose and microstomia, calcinosis and renal impairment.

Severe digital ulceration in such patients can be treated with infusion of a prostacyclin analogue such as iloprost. Prompt treatment is required to avoid gangrene and loss of digits. Drugs such as beta blockers and the oral contraceptive pill can exacerbate Raynauds phenomenon by causing vascular spasm and should therefore be avoided.

There is no indication for flucloxacillin if the ulcers are not infected.

Gloves and hand-warmers can be very helpful for patients with Raynauds phenomenon. Calcium channel blockers such as amlodipine and nifidepine cause vasodilation in peripheral arterioles are also used to treat Raynauds phenomenon. However, if there is severe ulceration admission for an iloprost infusion is required.



Next question >

# Raynaud's phenomenon \*

Raynaud's phenomenon is characterised by an exaggerated vasoconstrictive response of the digital arteries and cutaneous arteriole to the cold or emotional stress. It may be primary (Raynaud's disease) or secondary (Raynaud's phenomenon).

Raynaud's disease typically presents in young women (e.g. 30 years old) with bilateral symptoms.

Secondary causes of Raynaud's phenomenon

- connective tissue disorders
  - scleroderma (most common)
  - o rheumatoid arthritis
  - systemic lupus erythematosus
- leukaemia
- type I cryoglobulinaemia, cold agglutinins
- use of vibrating tools
- drugs: oral contraceptive pill, ergot
- cervical rib

Factors suggesting underlying connective tissue disease

- onset after 40 years
- unilateral symptoms
- rashes
- presence of autoantibodies
- features which may suggest rheumatoid arthritis or SLE, for example arthritis or recurrent miscarriages
- digital ulcers, calcinosis
- very rarely: chilblains

#### Management

- all patients with suspected secondary Raynaud's phenomenon should be referred to secondary care
- first-line: calcium channel blockers e.g. nifedipine
- IV prostacyclin (epoprostenol) infusions: effects may last several weeks/months







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Question 6 of 178





An 85-year-old man successfully completed treatment for community-acquired pneumonia. He has been medically stable for discharge but unfortunately is still an inpatient as he is awaiting a package of care to be sourced. His past medical history includes hypertension, ischaemic heart disease, type 2 diabetes and advanced chronic kidney disease (not for renal replacement therapy).

He complains of severe pain in his left foot. On examination, you note the metatarsophalangeal joint on the hallux is swollen, erythematous and hot to touch. No abnormality is detected on his right foot.

He is afebrile and scores 0 on his early warning score. His latest blood results are shown below:

Hb	120 g/L	Male: (135-180)	
Platelets	454 * 10 <sup>9</sup> /L	(150 - 400)	
WBC	10.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)	
Na <sup>+</sup>	137 mmol/L	(135 - 145)	
K <sup>+</sup>	4.9 mmol/L	(3.5 - 5.0)	
Urea	8.7 mmol/L	(2.0 - 7.0)	
Creatinine	525 µmol/L	(55 - 120)	
CRP	12 mg/L	(< 5)	

Given the likely diagnosis and his co-morbidities, what would be the most appropriate treatment to commence?

Prednisolone	
Colchicine	
Ibuprofen	
Febuxostat	
Allopurinol	

Submit answer

Reference ranges ✓

			Score: 0%		
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3 4	-				
5 <b>6</b>	-				

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Question 6 of 178



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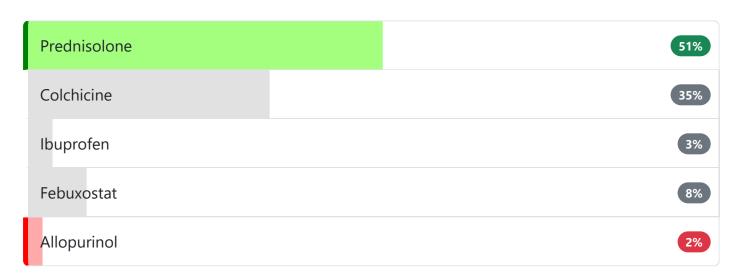
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Creatinine	525 µmol/L	(55 - 120)
CRP	12 mg/L	(< 5)

Given the likely diagnosis and his co-morbidities, what would be the most appropriate treatment to commence?



For gout, if NSAIDs and colchicine are contraindicated or not tolerated the next option is a steroid



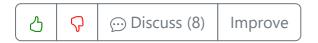
The patient is suffering from an acute attack of gout. The classical presentation is the 1st metatarsophalangeal joint being affected.

Whilst colchicine and NSAIDs such as ibuprofen are advocated by NICE as 1st line treatment for acute gout, they are contraindicated in this patient due to the severity of his CKD.

As a result, oral steroids such as prednisolone should be used.

Allopurinol is the 1st line treatment for the prevention of gout but has no benefit in an acute attack. However, if the patient is already on allopurinol, this can be continued throughout the acute flare.

Like allopurinol, febuxostat is a urate-lowering therapy used for the prevention of gout. It is used as a second-line treatment and is particularly useful in renal impairment where allopurinol is contraindicated.



Next question >

# Gout: management \*

Gout is a form of microcrystal synovitis caused by the deposition of monosodium urate monohydrate in the synovium. It is caused by chronic hyperuricaemia (uric acid >  $450 \mu mol/l$ )

# Acute management

- NSAIDs or colchicine are first-line
  - the maximum dose of NSAID should be prescribed until 1-2 days after the symptoms have settled
  - o gastroprotection (e.g. a proton pump inhibitor) may also be indicated
- colchicine
  - inhibits microtubule polymerization by binding to tubulin, interfering with mitosis. Also inhibits neutrophil motility and activity
  - has a slower onset of action
  - may be used with caution in renal impairment: the BNF advises to reduce the dose if eGFR is 10-50 ml/min and to avoid if eGFR < 10 ml/min BNF
  - the main side-effect is diarrhoea
- oral steroids may be considered if NSAIDs and colchicine are contraindicated.
  - o a dose of prednisolone 15mg/day is usually used
- another option is intra-articular steroid injection
- if the patient is already taking allopurinol it should be continued

Indications for urate-lowering therapy (ULT)

- the British Society of Rheumatology Guidelines now advocate offering urate-lowering therapy to all patients after their **first attack of gout**
- ULT is particularly recommended if:
  - > = 2 attacks in 12 months
  - o tophi
  - o renal disease
  - o uric acid renal stones
  - o prophylaxis if on cytotoxics or diuretics

# **Urate-lowering therapy**

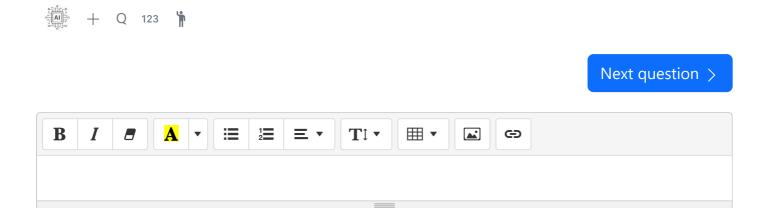
- it has traditionally been taught that urate-lowering therapy should not be started until 2 weeks after an acute attack, as starting too early may precipitate a further attack. The evidence base to support this however looks weak
- in 2017 the BSR updated their guidelines. They still support a delay in starting urate-lowering therapy because it is better for a patient to make long-term drug decisions whilst not in pain
  - the key passage is: 'Commencement of ULT is best delayed until inflammation has settled as ULT is better discussed when the patient is not in pain'
- allopurinol is first-line
  - $\circ$  initial dose of 100 mg od, with the dose titrated every few weeks to aim for a serum uric acid of < 360  $\mu$ mol/l
  - o a lower target uric acid level below 300 μmol/L may be considered for patients who have tophi, chronic gouty arthritis or continue to have ongoing frequent flares despite having a uric acid below 360 μmol/L
  - o a lower initial dose of allopurinol should be given if the patient has a reduced eGFR
  - colchicine cover should be considered when starting allopurinol. NSAIDs can be used if colchicine cannot be tolerated. The BSR guidelines suggest this may need to be continued for 6 months
- the second-line agent when allopurinol is not tolerated or ineffective is febuxostat (also a xanthine oxidase inhibitor)
- in refractory cases other agents may be tried:
  - uricase (urate oxidase) is an enzyme that catalyzes the conversion of urate to the degradation product allantoin. It is present in certain mammals but not humans
  - in patients who have persistent symptomatic and severe gout despite the adequate use of urate-lowering therapy, pegloticase (polyethylene glycol modified mammalian uricase) can achieve rapid control of hyperuricemia. It is given as an infusion once every two weeks

# Lifestyle modifications

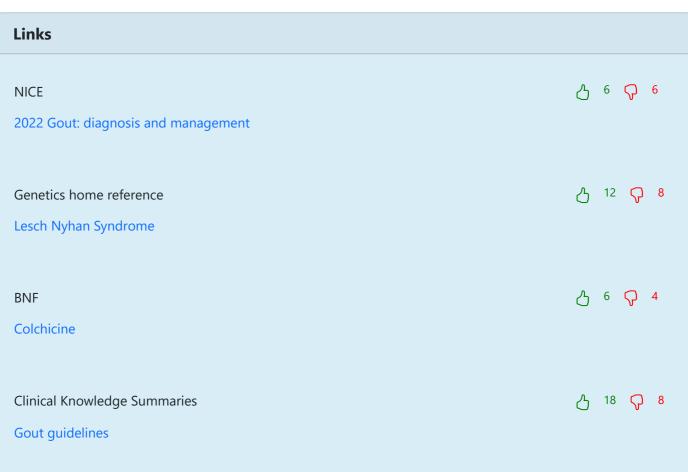
- reduce alcohol intake and avoid during an acute attack
- lose weight if obese
- avoid food high in purines e.g. Liver, kidneys, seafood, oily fish (mackerel, sardines) and yeast products

# Other points

- consideration should be given to stopping precipitating drugs (such as thiazides)
- losartan has a specific uricosuric action and may be particularly suitable for the many patients who have coexistent hypertension
- increased vitamin C intake (either supplements or through normal diet) may also decrease serum uric acid levels







Zero To Finals - YouTube

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Gout - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube

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Score: 18% 1 X 2 X X 3 4 X 5 X

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### Question 7 of 178





A 65-year-old man presents to an outpatient respiratory clinic with a 3-month history of weight loss, cough, weakness and intermittent haemoptysis. His past medical history includes type 2 diabetes and hypertension. He is on regular amlodipine, ramipril and metformin. He has smoked 15 cigarettes daily for approximately 40 years. He denies alcohol or recreational drug use.

On clinical examination, he appears underweight. His observations demonstrate a heart rate of 87 beats per minute, blood pressure 145/82 mmHg, respiratory rate 15/minute, oxygen saturations of 97% on room air and temperature of 36.7°C. Chest auscultation reveals a monophonic wheeze in the left upper lobe. His heart sounds are normal and there are no murmurs or peripheral oedema. There is no evidence of lymphadenopathy or organomegaly. His fingers are clubbed. Power is 3+/5 proximally in the upper and lower limbs. He finds it difficult to get up off his chair. Sensation is preserved, reflexes are normal and plantar reflexes are downgoing. There is no rash.

### Blood tests:

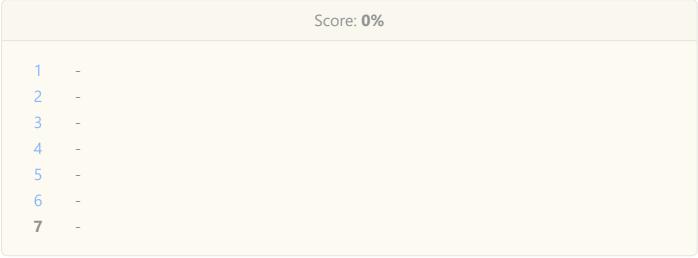
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Platelets	444 * 10 <sup>9</sup> /L	(150 - 400)
WBC	8.4 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	129 mmol/L	(135 - 145)
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Creatinine	111 μmol/L	(55 - 120)
CRP	8 mg/L	(< 5)
Creatine kinase	5891 U/L	(40-320)
TSH	5.9 miU/L	(0.2 - 5.5)
Free T4	11.1pmol/L	(10 - 24.5)

A chest x-ray demonstrates a coin lesion in the upper zone of the left lung.

What is the most appropriate initial treatment?

IV sodium bicarbonate	
Intravenous fluids	

Intravenous immunoglobulin	×
C Levothyroxine	×
<ul><li>Prednisolone</li></ul>	
Submit answer	
Reference ranges V	
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Question 7 of 178



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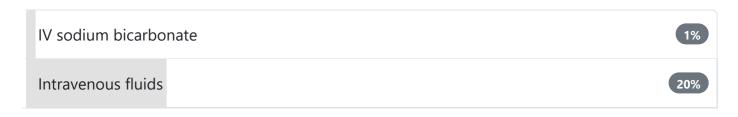
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What is the most appropriate initial treatment?



```
Intravenous immunoglobulin

Levothyroxine

Prednisolone

Malignancy + raised CK → ?polymyositis

Important for me Less important
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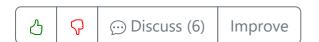
Prednisolone is correct. The history suggests a new diagnosis of lung cancer complicated by a cancer-associated myositis, given the significantly elevated creatine kinase and proximal weakness. The dose of prednisolone used to treat myositis is usually in the region of 1mg/kg with a slow taper over months. The co-existing malignancy will also need to be treated by usual means e.g. surgery, chemotherapy or radiotherapy depending on stage and grade.

IV sodium bicarbonate is incorrect. This is sometimes used to prevent acute kidney injury and treat metabolic acidosis in cases of rhabdomyolysis. However, given the clinical context of malignancy, raised creatine kinase and subacute weakness, this is not the correct answer.

Levothyroxine is not the right answer. The patient's thyroid function tests demonstrate mild subclinical hypothyroidism. Severe hypothyroidism can sometimes masquerade as a myositis but these results are not dramatic enough to give this picture.

Intravenous fluids are incorrect. This would be a sensible treatment if the diagnosis was rhabdomyolysis.

Intravenous immunoglobulin is not the right answer. This is a second-line treatment for those with refractory myositis.



Next question >

# Polymyositis \*

### Overview

- inflammatory disorder causing symmetrical, proximal muscle weakness
- thought to be a T-cell mediated cytotoxic process directed against muscle fibres
- may be idiopathic or associated with connective tissue disorders
- associated with malignancy

- dermatomyositis is a variant of the disease where skin manifestations are prominent, for example a purple (heliotrope) rash on the cheeks and eyelids
- typically affects middle-aged, female:male 3:1

### **Features**

- proximal muscle weakness +/- tenderness
- Raynaud's
- respiratory muscle weakness
- interstitial lung disease
  - o e.g. fibrosing alveolitis or organising pneumonia
  - o seen in around 20% of patients and indicates a poor prognosis
- dysphagia, dysphonia

# Investigations

- elevated creatine kinase
- other muscle enzymes (lactate dehydrogenase (LDH), aldolase, AST and ALT) are also elevated in 85-95% of patients
- EMG
- muscle biopsy
- anti-synthetase antibodies
  - anti-Jo-1 antibodies are seen in pattern of disease associated with lung involvement,
     Raynaud's and fever

# Management

- high-dose corticosteroids tapered as symptoms improve
- azathioprine may be used as a steroid-sparing agent



Next question >



# **Textbooks**

High-yield textbook

# Media



Polymyositis and Dermatomyositis in 3 Minutes

Townsend Teaching - YouTube









Understanding Myositis (Polymyositis and Dermatomyositis)

Zero To Finals - YouTube







Report broken media

Score: 18%

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Question 8 of 178





A 22-year-old woman presents to the emergency department with two weeks of sore throat, fever, rash and joint pains. She has no past medical history and does not take any regular medications. She does not smoke or drink alcohol. She has a family history of rheumatoid arthritis.

# Observations:

- Heart rate 89 beats per minute
- Respiratory rate 19/minute
- Oxygen saturations 96% on room air
- Temperature 39.1
- Blood pressure 124/77 mmHg

On examination, there is synovitis affecting the wrists bilaterally. There is a salmon-pink rash on her chest. There is evidence of pharyngitis. The examination is otherwise normal.

### Blood tests:

Hb	138 g/L	Male: (135-180) Female: (115 - 160)
Platelets	422 * 10 <sup>9</sup> /L	(150 - 400)
WBC	17.1 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	136 mmol/L	(135 - 145)
K <sup>+</sup>	4.2 mmol/L	(3.5 - 5.0)
Urea	5.4 mmol/L	(2.0 - 7.0)
Creatinine	101 µmol/L	(55 - 120)
Bilirubin	12 μmol/L	(3 - 17)
ALP	89 u/L	(30 - 100)
ALT	64 u/L	(3 - 40)
γGT	82 u/L	(8 - 60)
Albumin	36 g/L	(35 - 50)
CRP	52 mg/L	(<5)
Ferritin	2240 ng/mL	(20-250)

# Further testing:

Rheumatoid factor negative

Antinuclear antibody	negative
Blood cultures	negative

Plain radiography of the chest is unremarkable.

What is the most appropriate management?

Anakinra	
Corticosteroids	
Methotrexate	
NSAIDs	
Antibiotics	

Submit answer

Reference ranges  $\checkmark$ 

		Score: 0%
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Question 8 of 178



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# Further testing:

Rheumatoid factor negative

Antinuclear antibody	negative
Blood cultures	negative

Plain radiography of the chest is unremarkable.

What is the most appropriate management?



NSAIDs are the first line treatment for Still's disease, not steroids

Important for me Less important



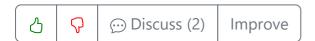
NSAIDs is the correct answer. This patient has Still's disease as evidenced by fever, characteristic rash, arthritis, deranged LFTs, hyperferritinaemia and negative testing for (antinuclear antibody) ANA and rheumatoid factor. She has no life-threatening organ dysfunction and therefore the initial management is with non-steroidal anti-inflammatory medications.

Corticosteroids is incorrect. This class of medication can be used if the condition is refractory to NSAID treatment or if there is more severe disease manifestations and organ dysfunction.

Methotrexate is incorrect. If the patient needs maintenance disease-modifying therapy, then this would be an option. However, it is not used to induce remission.

Antibiotics are incorrect. While the patient has a fever and raised inflammatory markers, the clinical picture is that of Still's disease, rather than infection.

Anakinra is incorrect. This is an interleukin (IL)-1 inhibitor used in the treatment of a variety of inflammatory conditions including Still's disease. It is used for severe cases that are refractory to initial treatment.



# Still's disease in adults \*

# **Epidemiology**

• has a bimodal age distribution - 15-25 yrs and 35-46 yrs

### **Features**

- arthralgia
- elevated serum ferritin
- rash: salmon-pink, maculopapular
- pyrexia
  - typically rises in the late afternoon/early evening in a daily pattern and accompanies a worsening of joint symptoms and rash
- lymphadenopathy
- rheumatoid factor (RF) and anti-nuclear antibody (ANA) negative

The diagnosis of Still's disease in adults can be challenging. The Yamaguchi criteria is the most widely used criteria and has a sensitivity of 93.5%.

# Management

- NSAIDs
  - should be used first-line to manage fever, joint pain and serositis
  - they should be trialled for at least a week before steroids are added.
- steroids
  - o may control symptoms but won't improve prognosis
- if symptoms persist, the use of methotrexate, IL-1 or anti-TNF therapy can be considered



Next question >



### **Textbooks**

High-yield textbook

Extended textbook

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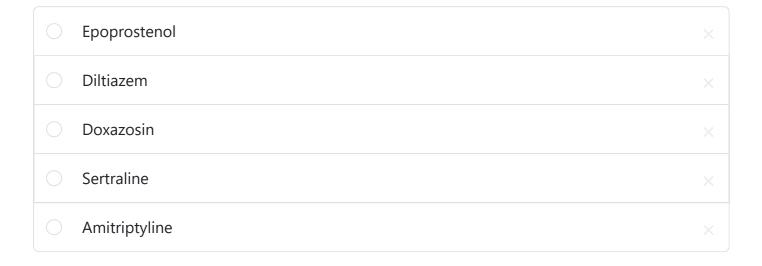




A 62-year-old lady with idiopathic Raynaud's comes to clinic concerned regarding painful, 'blue' fingers and areas that appear to be consistent with the beginning of ulceration. She has tried numerous over the counter medications, heating techniques and calcium channel blockers with no improvement. She is also known to suffer from hypertension for which she is on Losartan.

On examination you note prominent acrocyanosis with areas of skin discolouration and moderate digital ulceration. There is no sclerodactyly or telangiectasiae. Her blood pressure is: 105/60 mmHg.

What medication could be offered to this lady to help with her symptoms?



Submit answer

Reference ranges ∨

# Score: **0%**1 2 3 4 5 6 7 8 -

Back to top





Question 9 of 178





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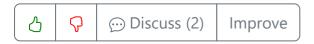


Epoprostenol (amongst other prostaglandins) can be used in the treatment of Raynaud's phenomenon

Important for me Less important



In Raynaud's phenomenon (whether idiopathic or not) optimisation of vasodilation is important. This is a lady however who has not tolerated the use of CCB. Given the presence of digital ulceration, sildenafil is the first line option for treatment. As this is not offered as a choice the correct decision would be the administration of IV prostanoids in the form of epoprostenol.



Next question >



Raynaud's phenomenon is characterised by an exaggerated vasoconstrictive response of the digital arteries and cutaneous arteriole to the cold or emotional stress. It may be primary (Raynaud's disease) or secondary (Raynaud's phenomenon).

Raynaud's disease typically presents in young women (e.g. 30 years old) with bilateral symptoms.

Secondary causes of Raynaud's phenomenon

- connective tissue disorders
  - scleroderma (most common)
  - o rheumatoid arthritis
  - systemic lupus erythematosus
- leukaemia
- type I cryoglobulinaemia, cold agglutinins
- use of vibrating tools
- drugs: oral contraceptive pill, ergot
- cervical rib

Factors suggesting underlying connective tissue disease

- onset after 40 years
- unilateral symptoms
- rashes
- presence of autoantibodies
- features which may suggest rheumatoid arthritis or SLE, for example arthritis or recurrent miscarriages
- digital ulcers, calcinosis
- very rarely: chilblains

# Management

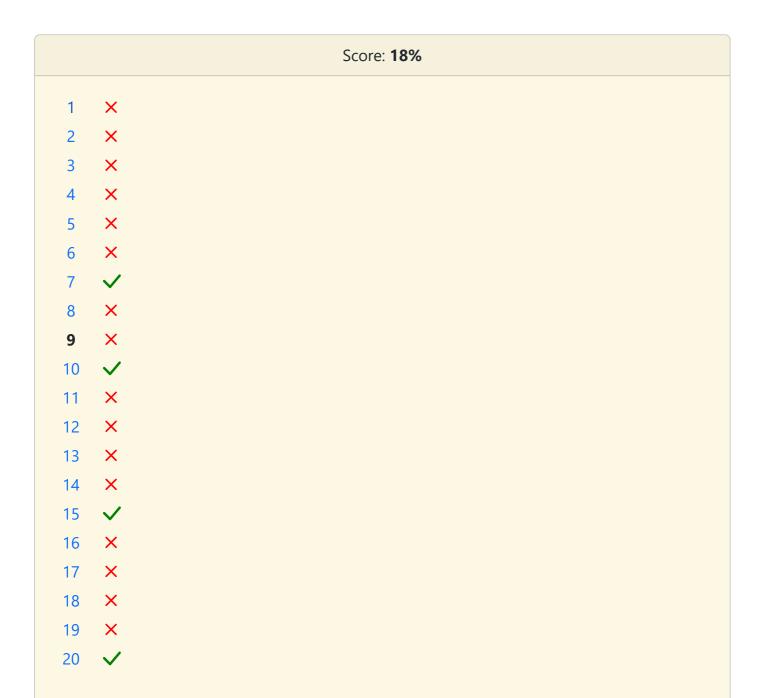
- all patients with suspected secondary Raynaud's phenomenon should be referred to secondary care
- first-line: calcium channel blockers e.g. nifedipine
- IV prostacyclin (epoprostenol) infusions: effects may last several weeks/months



Next question >







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### Question 10 of 178





A 69-year-old man attends the medical unit with an acutely hot and swollen joint. The swelling is limited to the first metatarsophalangeal joint **(MTPJ)** and is causing him severe discomfort. He states that he has had this problem before and had it successfully treated, but the medicine used caused him to have severe diarrhoea.

On examination, their first MTPJ is swollen, red and very tender to touch. There is limited movement and when they try to walk, it causes severe pain.

Their past medical history includes chronic kidney disease, gout, osteoarthritis and angina.

Blood tests taken at the time of admission:

Hb	140 g/L	Male: (135-180) Female: (115 - 160)
Platelets	300* 10 <sup>9</sup> /L	(150 - 400)
WBC	10.4 * 10 <sup>9</sup> /L	(4.0 - 11.0)

Na <sup>+</sup>	138 mmol/L	(135 - 145)
K <sup>+</sup>	4.8 mmol/L	(3.5 - 5.0)
Urea	14 mmol/L	(2.0 - 7.0)
Creatinine	230 µmol/L	(55 - 120)
CRP	32 mg/L	(< 5)

You suspect the diagnosis is an acute flare of gout.

What is the most appropriate treatment?

Colchicine	
Febuxostat	
IM steroid	
Ibuprofen	
Oral prednisolone	

# Submit answer

Reference ranges  $\vee$ 

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Question 10 of 178



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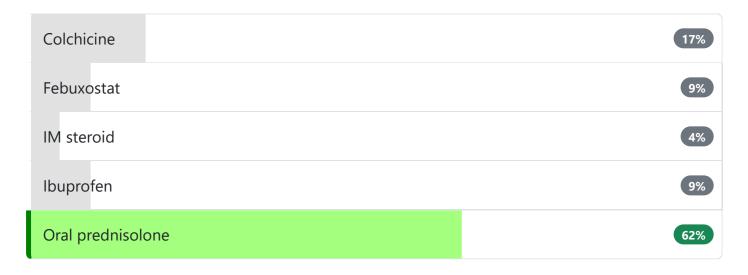
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CRP	32 mg/L	(< 5)

You suspect the diagnosis is an acute flare of gout.

What is the most appropriate treatment?



For gout, if NSAIDs and colchicine are contraindicated or not tolerated the next option is a steroid

Important for me Less important



The answer is oral prednisolone as the most appropriate treatment for this man. He has gout affecting one small joint with relative contraindications to NSAIDS and colchicine (asthma, previous intolerance of colchicine and renal disease).

Febuxostat is not an appropriate choice as this a gout prophylaxis medication.

An alternative treatment would be intra-articular injection of steroid in this case but not an IM steroid injection.



Next question

# Gout: management \*

Gout is a form of microcrystal synovitis caused by the deposition of monosodium urate monohydrate in the synovium. It is caused by chronic hyperuricaemia (uric acid > 450 µmol/l)

# Acute management

- NSAIDs or colchicine are first-line
  - the maximum dose of NSAID should be prescribed until 1-2 days after the symptoms have settled
  - o gastroprotection (e.g. a proton pump inhibitor) may also be indicated
- colchicine
  - inhibits microtubule polymerization by binding to tubulin, interfering with mitosis. Also inhibits neutrophil motility and activity
  - has a slower onset of action
  - may be used with caution in renal impairment: the BNF advises to reduce the dose if eGFR is 10-50 ml/min and to avoid if eGFR < 10 ml/min (BNF)
  - o the main side-effect is diarrhoea
- oral steroids may be considered if NSAIDs and colchicine are contraindicated.
  - o a dose of prednisolone 15mg/day is usually used
- another option is intra-articular steroid injection
- if the patient is already taking allopurinol it should be continued

- the British Society of Rheumatology Guidelines now advocate offering urate-lowering therapy to all patients after their **first attack of gout**
- ULT is particularly recommended if:
  - > = 2 attacks in 12 months
  - o tophi
  - o renal disease
  - uric acid renal stones
  - prophylaxis if on cytotoxics or diuretics

# **Urate-lowering therapy**

- it has traditionally been taught that urate-lowering therapy should not be started until 2 weeks after an acute attack, as starting too early may precipitate a further attack. The evidence base to support this however looks weak
- in 2017 the BSR updated their guidelines. They still support a delay in starting urate-lowering therapy because it is better for a patient to make long-term drug decisions whilst not in pain
  - the key passage is: 'Commencement of ULT is best delayed until inflammation has settled as ULT is better discussed when the patient is not in pain'
- allopurinol is first-line
  - $\circ$  initial dose of 100 mg od, with the dose titrated every few weeks to aim for a serum uric acid of < 360  $\mu$ mol/l
  - $\circ$  a lower target uric acid level below 300 µmol/L may be considered for patients who have tophi, chronic gouty arthritis or continue to have ongoing frequent flares despite having a uric acid below 360 µmol/L
  - o a lower initial dose of allopurinol should be given if the patient has a reduced eGFR
  - colchicine cover should be considered when starting allopurinol. NSAIDs can be used if colchicine cannot be tolerated. The BSR guidelines suggest this may need to be continued for 6 months
- the second-line agent when allopurinol is not tolerated or ineffective is febuxostat (also a xanthine oxidase inhibitor)
- in refractory cases other agents may be tried:
  - uricase (urate oxidase) is an enzyme that catalyzes the conversion of urate to the degradation product allantoin. It is present in certain mammals but not humans
  - in patients who have persistent symptomatic and severe gout despite the adequate use
    of urate-lowering therapy, pegloticase (polyethylene glycol modified mammalian uricase)
    can achieve rapid control of hyperuricemia. It is given as an infusion once every two
    weeks

# Lifestyle modifications

- reduce alcohol intake and avoid during an acute attack
- lose weight if obese
- avoid food high in purines e.g. Liver, kidneys, seafood, oily fish (mackerel, sardines) and yeast products

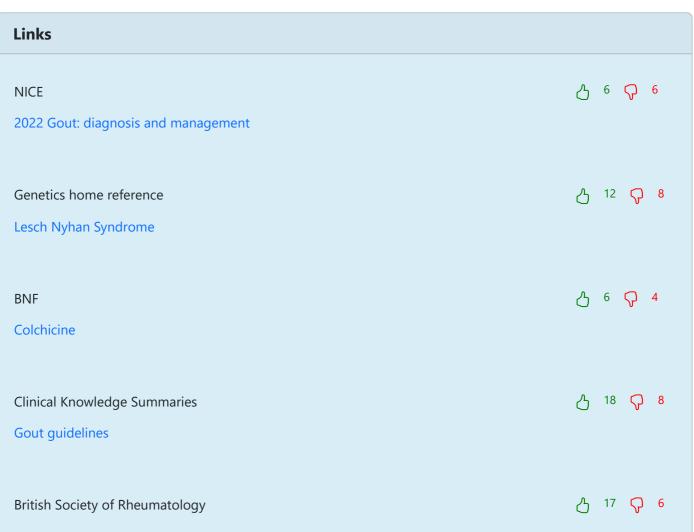
- consideration should be given to stopping precipitating drugs (such as thiazides)
- losartan has a specific uricosuric action and may be particularly suitable for the many patients who have coexistent hypertension
- increased vitamin C intake (either supplements or through normal diet) may also decrease serum uric acid levels



Next question >







Suggest link Report broken link

# Media



# Gout

Rhesus Medicine - YouTube





### Gout

Zero To Finals - YouTube





Gout - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube



Report broken media

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#### Question 11 of 178

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A 25-year-old woman is admitted to the hospital with recurrent fevers. She also complains of joint pain. There is no recent history of foreign travel, and she denies any recreational drug use. Observations include a temperature of 39.2°C, pulse rate of 92, blood pressure of 124/72 mmHg, respiratory rate of 18/min, and oxygen saturation of 97% on room air. On examination, there is a salmon-pink rash on her trunk that feels bumpy to the touch. There is also mild cervical lymphadenopathy. Blood results are as follows:

Hb	129 g/L	Male: (135 - 180) Female: (115 - 160)
Platelets	244 * 10 <sup>9</sup> /L	(150 - 400)
WBC	12.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Bilirubin	15 μmol/L	(3 - 17)
ALP	120 u/L	(30 - 100)
ALT	50 u/L	(3 - 40)
γGT	72 u/L	(8 - 60)
Albumin	38 g/L	(35 - 50)

ESR	35 mm/hr	Men: (<22) Women: (<17)
Prothrombin time (PT)	12 secs	(10-14 secs)
Activated partial thromboplastin time (APTT)	27 secs	(25-35 secs)
Fibrinogen	2 g/L	(2 - 4)
Ferritin	2100 ng/mL	(20 - 230)
Vitamin B12	450 ng/L	(200 - 900)
Folate	4.2 nmol/L	(>3.0)
Reticulocytes	0.7%	(0.5 - 1.5)

What is the most appropriate first-line treatment given the probable diagnosis?

Azathioprine	
Ciprofloxacin	
Ibuprofen	
Prednisolone	

Rituximab			

# Submit answer

Reference ranges ✓

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Question 11 of 178



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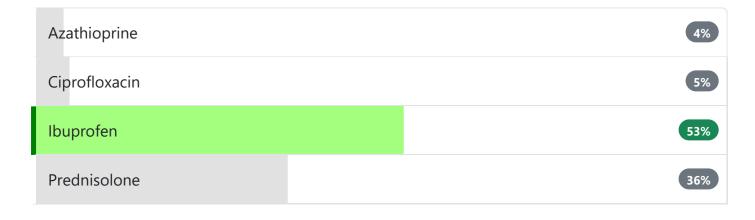


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Reticulocytes	0.7%	(0.5 - 1.5)

What is the most appropriate first-line treatment given the probable diagnosis?



#### NSAIDs are the first line treatment for Still's disease, not steroids

Important for me Less important



This patient is displaying the classic triad of fevers, joint pain, and a salmon-colored bumpy rash. The diagnosis of adult-onset Still's disease (AOSD) is considered a diagnosis of exclusion and is normally a clinical one. The majority of patients will have a very high ferritin level and a high ESR. Derangement in liver function tests is also common. First-line treatment involves NSAIDs such as ibuprofen or naproxen.

Immunosuppressive therapy can then be used to control the disease and induce remission in severe disease. Options include steroids such as prednisolone.

Ciprofloxacin is an antibiotic that can be used to treat several infections including typhoid fever, which can also present with a salmon-pink rash. Typhoid fever is incredibly unlikely given the negative travel history.

Azathioprine is another immunosuppressive commonly used in rheumatoid arthritis, granulomatosis with polyangiitis, and other conditions. It can also be given post-kidney transplant to prevent rejection.

Rituximab is a monoclonal antibody used to treat many autoimmune diseases. It has been used in drug-resistant AOSD.



Next question >

### Still's disease in adults \*

#### Epidemiology

• has a bimodal age distribution - 15-25 yrs and 35-46 yrs

#### **Features**

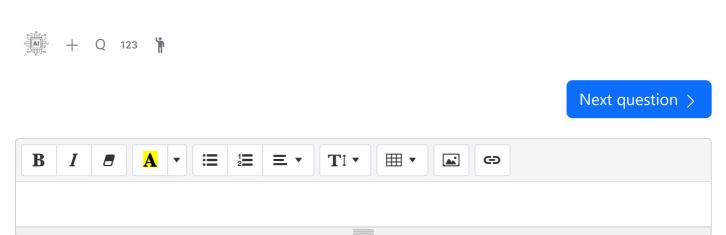
- arthralgia
- elevated serum ferritin
- rash: salmon-pink, maculopapular
- pyrexia

- typically rises in the late afternoon/early evening in a daily pattern and accompanies a worsening of joint symptoms and rash
- lymphadenopathy
- rheumatoid factor (RF) and anti-nuclear antibody (ANA) negative

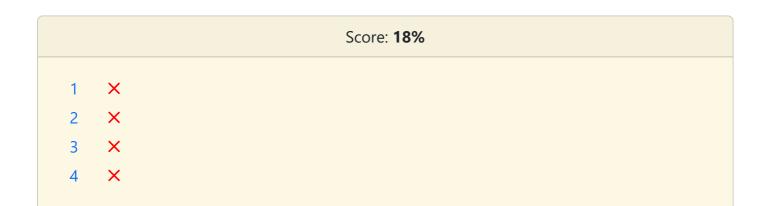
The diagnosis of Still's disease in adults can be challenging. The Yamaguchi criteria is the most widely used criteria and has a sensitivity of 93.5%.

#### Management

- NSAIDs
  - should be used first-line to manage fever, joint pain and serositis
  - they should be trialled for at least a week before steroids are added.
- steroids
  - o may control symptoms but won't improve prognosis
- if symptoms persist, the use of methotrexate, IL-1 or anti-TNF therapy can be considered







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Question 12 of 178





A 47-year-old man has asked for a review of his right elbow due to pain and a restriction in the range of movement. He denies trauma or a recent injury.

There is no past medical history and he works as a plumber, does not smoke, drinks 35 units of alcohol per week, and is not keen on exercise.

On examination of his elbow, there is no evidence of effusion or swelling. Pain is exacerbated during wrist extension and supination whilst the elbow is extended. There is no motor or sensory deficit.

What is the likely diagnosis?

Cubital tunnel syndrome	
Lateral epicondylitis	
Medial epicondylitis	
Olecranon bursitis	
Triceps tendonitis	

Submit answer

Reference ranges ∨

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Question 12 of 178



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What is the likely diagnosis?



Lateral epicondylitis: worse on resisted wrist extension/suppination whilst elbow extended

| Important for me | Less important |



Examination findings of pain or weakness being exacerbated when the wrist is extended or supinated during elbow extension are indicative of **lateral epicondylitis**. Also known as tennis elbow, lateral epicondylitis can be caused or exacerbated by repetitive occupational or recreational movements. His career as a plumber is likely to be the cause.

**Cubital tunnel syndrome** is caused by ulnar nerve entrapment and commonly causes medial elbow pain with paraesthesia in the ulnar nerve distribution making this answer incorrect.

**Medial epicondylitis**, or golfer's elbow, is also secondary to repetitive occupational or recreational activity. Pain is exacerbated with resisted wrist flexion and pronation, the opposite of lateral epicondylitis.

**Olecranon bursitis** typically presents as a boggy, non-tender swelling over the olecranon following trauma. This is incorrect as clinical examination showed no evidence of swelling or effusion and there was no history of trauma.

**Triceps tendonitis** presents as pain in the posterior aspect of the elbow which is exacerbated on resisted extension. There is rarely worsening pain with supination making this answer incorrect.



Next question >

# Lateral epicondylitis \*

Lateral epicondylitis typically follows unaccustomed activity such as house painting or playing tennis ('tennis elbow'). It is most common in people aged 45-55 years and typically affects the dominant arm.

#### **Features**

- pain and tenderness localised to the lateral epicondyle
- pain worse on wrist extension against resistance with the elbow extended or supination of the forearm with the elbow extended
- episodes typically last between 6 months and 2 years. Patients tend to have acute pain for 6-12 weeks

#### Management options

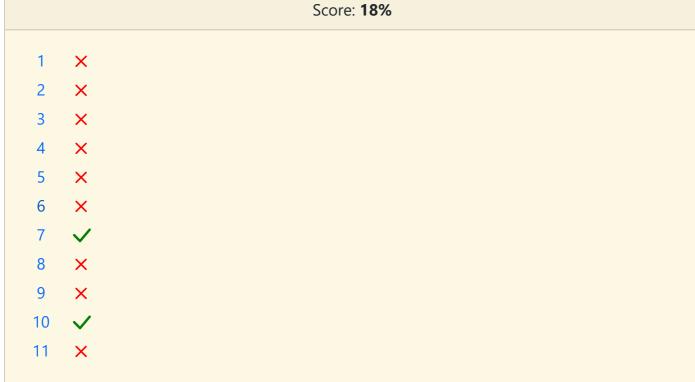
- advice on avoiding muscle overload
- simple analgesia
- steroid injection
- physiotherapy



Next question >



# **Textbooks** High-yield textbook Extended textbook Links 占 11 ♀ 6 Clinical Knowledge Summaries Tennis elbow Suggest link Report broken link Media TENNIS&GOLFERS Understanding Tennis Elbow and Golfer's Elbow (Lateral & Medial Epicondylitis) Rhesus Medicine - YouTube Report broken media Score: 18%



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A 7-year-old girl who has recently emigrated from Turkey is brought to the Emergency Department with pain on walking and multiple swellings over her head. She also complains of persistent headaches which are now quite distracting and causing her to miss school. This symptoms have been getting gradually worse for the past few weeks.

She has a past medical history of eczema and asthma which is well controlled with a salbutamol inhaler as required. There is no family history of similar problems.

On examination a number of soft tissue swellings are noted on the scalp. She also has non-specific tenderness over the proximal part of the left femur.

#### A skull x-ray is requested:



#### What is the most likely diagnosis?

Multiple myeloma	
Langerhans cell histiocytosis	
Neutrofibromatosis	
Systemic mastocytosis	
Wiskott-Aldrich syndrome	

Submit answer

Reference ranges ✓

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Question 13 of 178





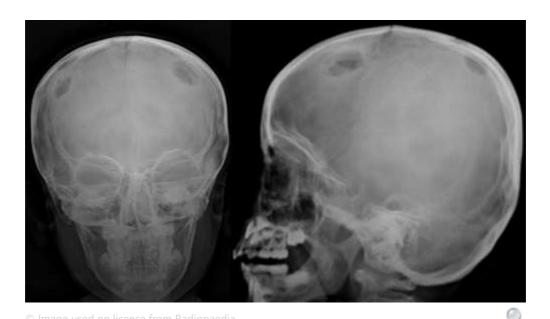


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On examination a number of soft tissue swellings are noted on the scalp. She also has non-specific tenderness over the proximal part of the left femur.

#### A skull x-ray is requested:



#### What is the most likely diagnosis?

Multiple myeloma	5%
Langerhans cell histiocytosis	66%
Neutrofibromatosis	4%
Systemic mastocytosis	11%
Wiskott-Aldrich syndrome	15%



The age of the patient combined with 'punched out' osteolytic skull lesions make a diagnosis of Langerhans cell histiocytosis likely.

Systemic mastocytosis results from a neoplastic proliferation of mast cells and is associated with urticaria pigmentosa and flushing.



Next question >

# Langerhans cell histiocytosis \*

Langerhans cell histiocytosis is a rare disorder characterised by the proliferation of Langerhans cells, which are specialised dendritic cells that normally function to present antigen to T lymphocytes. The disease can affect multiple organs, including the bones, skin, lungs, and endocrine system. It is notable for its variable clinical presentation, ranging from isolated bone lesions to multisystem disease.

#### **Features**

- bone pain, typically in the skull or proximal femur
- cutaneous nodules
- pituitary involvement: leads to diabetes insipidus due to pituitary stalk involvement
- pulmonary involvement: More common in adults, presenting with dyspnoea, cough, and chest pain
- recurrent otitis media/mastoiditis
- tennis racket-shaped Birbeck granules on electromicroscopy

#### Diagnosis:

- biopsy: confirmation is through biopsy showing Langerhans cells with characteristic grooved nuclei and positive staining for CD1a and S100 protein
- imaging: radiographs and MRI for bone lesions; CT may be used for chest and abdominal involvement

#### Treatment:

- localized disease: surgical resection or limited radiotherapy for isolated lesions.
- multisystem disease: systemic therapy including steroids, chemotherapy (e.g., vinblastine, cytarabine), and targeted therapies for refractory cases.
- supportive care: management of diabetes insipidus, pain control, and treatment of secondary infections.



© Image used on license from Radiopaedia

Young girl with multiple well defined 'punched out' osteolytic lesions with scalloped edges (geographic skull) are seen in the bilateral parietal regions. The lesions have a characteristic bevelled edge.



Next question >



#### **Textbooks**

High-yield textbook

Extended textbook

#### Media



Langerhans cell histiocytosis

Report broken media

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Question 14 of 178





62-year-old woman complains of knee pain. She has struggled with pain for several years. She finds that it gets worse through the day and is relieved by resting. She does not normally come and see doctors but the pain has gotten to the point where she would like additional treatment. She is not keen on surgery as of yet. She is known to have osteoarthritis. She has been taking paracetamol but not tried any other medication. What is the most appropriate strategy to further help relieve her pain?

Codeine	
Oral NSAIDs	
Topical NSAIDs	
Intra-articular steroid injection	
Oral morphine	

Submit answer

Reference ranges ∨

# Score: **0%**1 2 3 4 5 6 7 8 9 10 11 -

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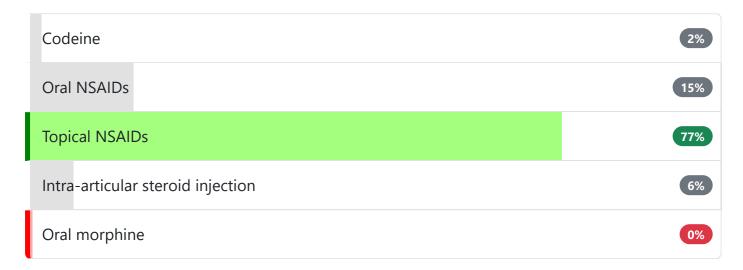


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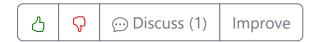
62-year-old woman complains of knee pain. She has struggled with pain for several years. She finds that it gets worse through the day and is relieved by resting. She does not normally come and see doctors but the pain has gotten to the point where she would like additional treatment. She is not keen on surgery as of yet. She is known to have osteoarthritis. She has been taking paracetamol but not tried any other medication. What is the most appropriate strategy to further help relieve her pain?

X





The correct answer is topical NSAIDs. This is a case of managing pain in osteoarthritis. As she has knee osteoarthritis the first line of treatment can include paracetamol and topical NSAIDs. Topical NSAIDs are only appropriate for osteoarthritis of the hands and knees. Second line treatment includes oral NSAIDs, codeine, capsaicin cream and intra-articular corticosteroids.



Next question >

# Osteoarthritis: management \*

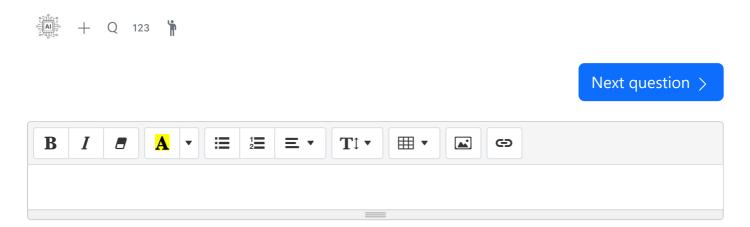
NICE updated its guidelines on the management of osteoarthritis (OA) in 2022

- all patients should be offered help with weight loss, given advice about local muscle strengthening exercises and general aerobic fitness
- topical NSAIDs are first-line analgesics. Topical NSAIDs may be particularly beneficial for patients with OA of the knee or hand
- second-line treatment is oral NSAIDs
  - a proton pump inhibitor should be co-prescribed with NSAIDs
  - o these drugs should be avoided if the patient takes aspirin

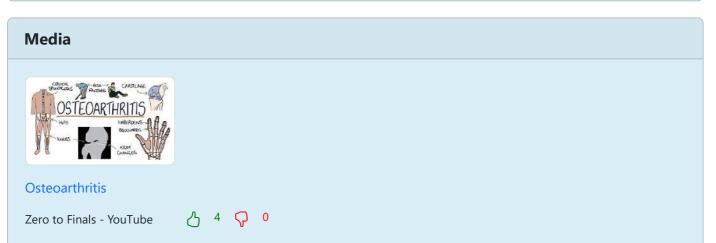
- NICE recommend we do not offer paracetamol or weak opioids, unless: NICE
  - o they are only used infrequently for short-term pain relief and
  - o all other pharmacological treatments are contraindicated, not tolerated or ineffective
- glucosamine and strong opioids are not recommended

**Textbooks** 

- non-pharmacological treatment options include walking aids for knee and hip OA
- intra-articular steroid injections may be tried if standard pharmacological treatment is ineffective
  - o patients should be aware that they only provide short-term relief (2-10 weeks)
- if conservative methods fail then refer for consideration of joint replacement









## Osteoarthritis Explained Clearly

Rhesus Medicine - YouTube 6 0 0 0

25







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#### Score: **18%** 1 X 2 X 3 X 4 × × 5 6 × 7 × 8 × 9 10 × 11 12 X 13 × 14 15 X 16 × 17 × 18 19 × 20 21 X 22 X 23 X 24 X

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#### Question 15 of 178

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A 72-year-old man presents by blue light ambulance to the emergency department. He has been increasingly drowsy over the past 4 days and has not been out of bed for the past 48 hours. His wife reports two episodes of sweating and a high temperature during this period. He has been admitted three times in the last 9 months with urinary tract infections and is awaiting a transurethral resection of his prostate for benign prostatic hypertrophy. His other past medical history includes type 2 diabetes mellitus, diagnosed 4 years ago, and rheumatoid arthritis diagnosed 16 years ago. His wife tells you that he appears to be prone to infections over the past few years. He normally walks with a stick but his exercise tolerance has been decreasing since doctors told him that he had 'scarring of his lungs from his rheumatoid.' On examination, he is sleepy but easily rousable and orientated to time and place. He is cool peripherally, with dry mucous membranes and JVP +1cm above the angle of Louis. Blood pressure measures 82/55mmHg, heart rate is 105/minute. You note conjunctival pallor, bilateral ulnar deviation of his hands, an inflamed second MTP joint and nodules beneath both elbows. Auscultation of the chest demonstrates bibasal inspiratory fine crackles. Abdominal examination demonstrates a 2cm liver edge and a 13cm spleen. Neurological examination is unremarkable. Routine blood tests and blood cultures are taken. What is the most appropriate treatment?

Intravenous antibiotics as per local guidelines for severe community acquired pneumonia $\times$	
Intravenous antibiotics as per local guidelines for urosepsis ×	

Oral antibiotics as per local guidelines for mild-moderate community acquired pneumonia

Intravenous antibiotics as per local guidelines for intra-abdominal sepsis

Intravenous antibiotic as per local guidelines for neutropenic sepsis

A second and per second general and a second per second

Submit answer

Reference ranges ✓

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Question 15 of 178



A 72-year-old man presents by blue light ambulance to the emergency department. He has been increasingly drowsy over the past 4 days and has not been out of bed for the past 48 hours. His wife reports two episodes of sweating and a high temperature during this period. He has been admitted three times in the last 9 months with urinary tract infections and is awaiting a transurethral resection of his prostate for benign prostatic hypertrophy. His other past medical history includes type 2 diabetes mellitus, diagnosed 4 years ago, and rheumatoid arthritis diagnosed 16 years ago. His wife tells you that he appears to be prone to infections over the past few years. He normally walks with a stick but his exercise tolerance has been decreasing since doctors told him that he had 'scarring of his lungs from his rheumatoid.' On examination, he is sleepy but easily rousable and orientated to time and place. He is cool peripherally, with dry mucous membranes and JVP +1cm above the angle of Louis. Blood pressure measures 82/55mmHg, heart rate is 105/minute. You note conjunctival pallor, bilateral ulnar deviation of his hands, an inflamed second MTP joint and nodules beneath both elbows. Auscultation of the chest demonstrates bibasal inspiratory fine crackles. Abdominal examination demonstrates a 2cm liver edge and a 13cm spleen. Neurological examination is unremarkable. Routine blood tests and blood cultures are taken. What is the most appropriate treatment?

Oral antibiotics as per local guidelines for mild-moderate community acquired pneumonia 1% Intravenous antibiotics as per local guidelines for severe community acquired pneumonia (9%) Intravenous antibiotics as per local guidelines for urosepsis 19% Intravenous antibiotics as per local guidelines for intra-abdominal sepsis 3% Intravenous antibiotic as per local guidelines for neutropenic sepsis 67%



The patient is known to have rheumatoid arthritis with multiple extra-articular features. He is also clinically septic and intravascularly dehydrated. The proneness to infections must raise suspicions of neutropenia, combining with RA with extra-articular features, splenomegaly to produce the classic triad of Felty's syndrome. Although he has had multiple episodes of previous UTIs, the history is unclear here. Neutropenic sepsis without a clear source of infection must be treated with intravenous antibiotics as per local guidelines.





A wide variety of extra-articular complications occur in patients with rheumatoid arthritis (RA):

- respiratory: pulmonary fibrosis, pleural effusion, pulmonary nodules, bronchiolitis obliterans, methotrexate pneumonitis, pleurisy
- ocular: keratoconjunctivitis sicca (most common), episcleritis, scleritis, corneal ulceration, keratitis, steroid-induced cataracts, chloroquine retinopathy
- osteoporosis
- ischaemic heart disease: RA carries a similar risk to type 2 diabetes mellitus
- increased risk of infections
- depression

#### Less common

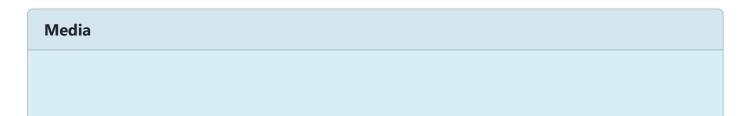
- Felty's syndrome (RA + splenomegaly + low white cell count)
- amyloidosis



Next question >









#### Rheumatoid arthritis

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#### Score: 18% X 1 2 X 3 X 4 × X 5 6 X 7 8 X X 9 10 11 X 12 X 13 × 14 × 15 16 X 17 × 18 × X 19 20 21 × 22 × 23 X 24 X 25 🗸

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Question 16 of 178





A 62-year-old lady is seen in the rheumatology clinic. She was diagnosed with rheumatoid arthritis 16 years ago. Her symptoms were relatively well controlled with a combination of methotrexate 20mg once per week, folic acid 5mg once per week and azathioprine 100mg once per day until the last few months when she complained of increasing joint pain with stiffness. Since then her methotrexate dose was gradually titrated to the current dose of 25mg per week. She reported that her joints were less painful and stiff in the morning. Unfortunately, she was also complained of increasing tiredness with an increasing quantity of respiratory tract infections, requiring antibiotics twice in the last six months. She also noted that she bruised more easily of late.

Examination revealed a slender 62-year-old systemically well lady. She was haemodynamically normal and afebrile. Cardiovascular and respiratory examinations were unremarkable, and abdominal examination revealed a mass arising from the left upper quadrant. Clinical examination of her joints revealed no evidence of synovitis or swelling.

Routine blood investigations prior to attending clinic were as follows:

Hb	115 g/l
MCV	84 fl
Platelets	82 * 10 <sup>9</sup> /I
WBC	3.5 * 10 <sup>9</sup> /l
Neutrophils	1.6 * 10 <sup>9</sup> /I
Lymphocytes	1.0 * 10 <sup>9</sup> /l
Eosinophils	0.9 * 10 <sup>9</sup> /l

Na <sup>+</sup>	141 mmol/l
K <sup>+</sup>	3.9 mmol/l
Urea	7.0 mmol/l
Creatinine	81 µmol/l

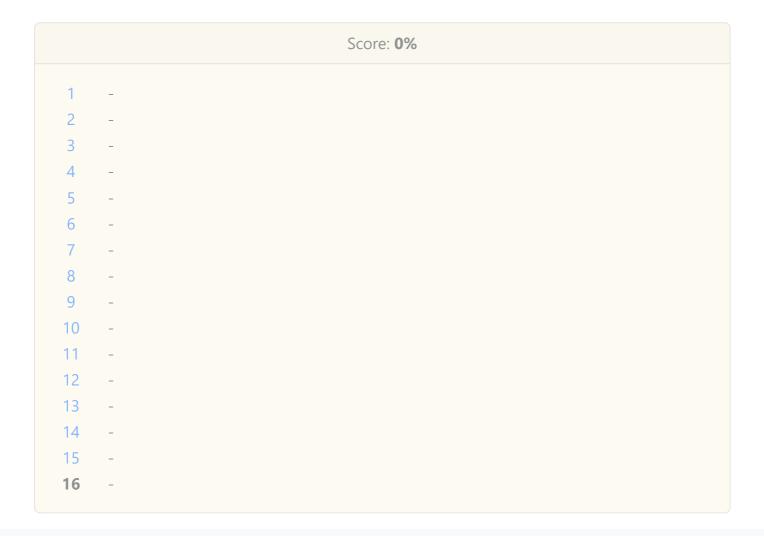
Bilirubin	12 µmol/l
ALP	99 u/l
ALT	13 u/l
Albumin	39 g/l

What is the single most likely cause of the clinical and haematological abnormalities?

	Myelodysplastic syndrome	
0	Chronic lymphocytic leukaemia	
0	Marrow aplasia secondary to drug therapy	
0	Felty's syndrome	
	Myelodysplasia	

# Submit answer

Reference ranges  $\vee$ 







Question 16 of 178







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Urea	7.0 mmol/l
Creatinine	81 µmol/l

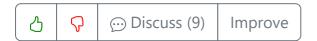
Bilirubin	12 µmol/l
ALP	99 u/l
ALT	13 u/l
Albumin	39 g/l

What is the single most likely cause of the clinical and haematological abnormalities?

Myelodysplastic syndrome					
Chronic lymphocytic leukaemia					
Marrow aplasia secondary to drug therapy	28%				
Felty's syndrome	66%				
Myelodysplasia	2%				



Felty's syndrome is a complication of Rheumatoid Arthritis (RA). It consists of a combination of rheumatoid arthritis, neutropaenia and splenomegaly, and tends to affect RA of longstanding duration. The main differential diagnosis is drug-induced marrow aplasia; however, this would not easily account for the presence of splenomegaly.



Next question >

### Rheumatoid arthritis: complications \*

A wide variety of extra-articular complications occur in patients with rheumatoid arthritis (RA):

- respiratory: pulmonary fibrosis, pleural effusion, pulmonary nodules, bronchiolitis obliterans, methotrexate pneumonitis, pleurisy
- ocular: keratoconjunctivitis sicca (most common), episcleritis, scleritis, corneal ulceration, keratitis, steroid-induced cataracts, chloroquine retinopathy
- osteoporosis
- ischaemic heart disease: RA carries a similar risk to type 2 diabetes mellitus
- increased risk of infections
- depression

### Less common

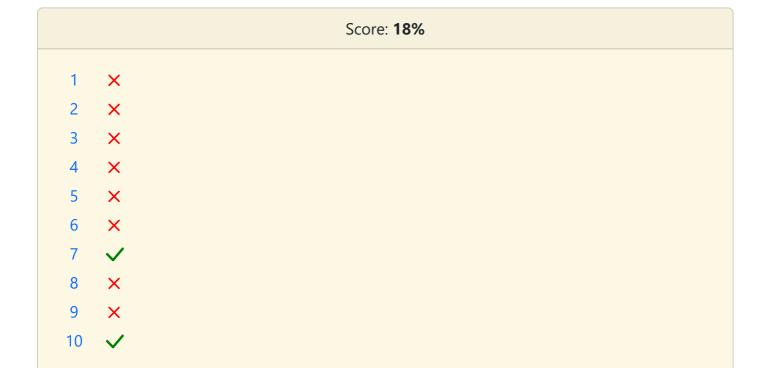
- Felty's syndrome (RA + splenomegaly + low white cell count)
- amyloidosis





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### Question 17 of 178

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A 74-year-old female was admitted to the medical ward initially for the treatment of a CURB = 4 community-acquired pneumonia. She is now awaiting discharge but since her illness, she has not returned to her pre-morbid state. Her past medical history includes two previous myocardial infarctions, hypertension, type 2 diabetes mellitus, duodenal ulcer and obesity. In addition, the physiotherapists report significant right knee pain to be contributing to poor mobility. On questioning, the patient reports that the pain is chronic and has been progressively worsening for about 3 years.

Her GP had sent her for two X-rays previously that demonstrated cartilage loss and osteophyte formation, with a reduction in joint space. On examination, you note significant crepitus in the right knee, with reduced range of movements in flexion and extension. You also note bony outgrowths in the proximal interphalangeal joints of her second and third digits of her right hand. She had successfully lost 9kg in weight and had previously taken 1g paracetamol four times a day regularly but neither measure seemed to help her pain.

What is the most appropriate next step?

Increase 500mg paracetamol as required	
Oral ibuprofen	
Topical diclofenac	
Oramorph as required	
Glucosamine	

Submit answer

Reference ranges ✓

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Question 17 of 178



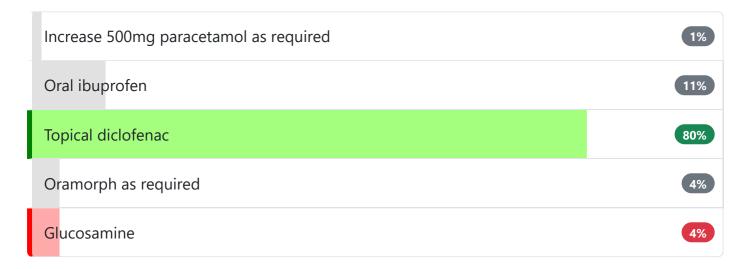




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What is the most appropriate next step?





The patient describes osteoarthritic symptoms that have persisted despite non-pharmacological therapies (weight loss) and paracetamol on a regular basis. Her past medical history of a duodenal ulcer should make you wary of oral NSAIDs while her previous MIs indicate selective COX-2 inhibitors should be used with caution. Opioids are not the second line therapies for osteoarthritis. The evidence for glucosamine is limited and is not recommended for use in the NHS. Topical NSAIDs such as diclofenac or topical capsaicin as an adjunct are reasonable options in this setting. A 3rd line possibility if topical NSAIDs are not efficacious and in this case, where oral NSAIDs are contraindicated, are intraarticular steroid injections, which have been demonstrated to produce significant symptomatic improvements in the knee joint when compared to placebo (although evidence is weaker for other joints). Interestingly, an inflammatory element is not required for

symptomatic benefits.

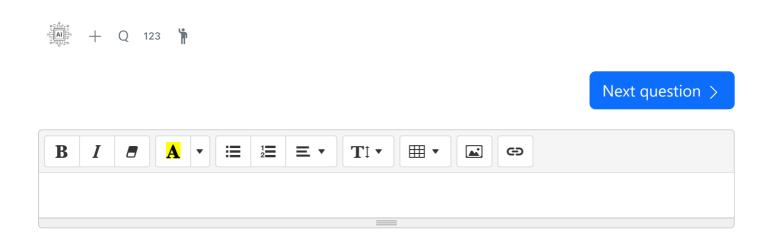


Next question >

### Osteoarthritis: management \*

NICE updated its guidelines on the management of osteoarthritis (OA) in 2022

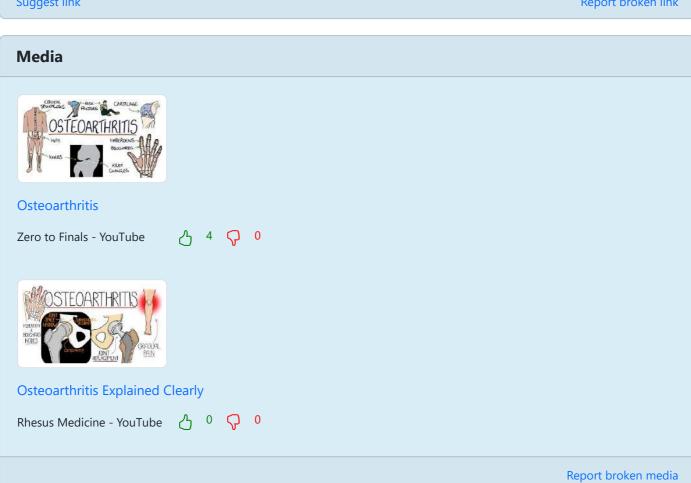
- all patients should be offered help with weight loss, given advice about local muscle strengthening exercises and general aerobic fitness
- topical NSAIDs are first-line analgesics. Topical NSAIDs may be particularly beneficial for patients with OA of the knee or hand NICE
- second-line treatment is oral NSAIDs
  - o a proton pump inhibitor should be co-prescribed with NSAIDs
  - these drugs should be avoided if the patient takes aspirin
- NICE recommend we do not offer paracetamol or weak opioids, unless: NICE
  - they are only used infrequently for short-term pain relief and
  - o all other pharmacological treatments are contraindicated, not tolerated or ineffective
- glucosamine and strong opioids are not recommended
- non-pharmacological treatment options include walking aids for knee and hip OA
- intra-articular steroid injections may be tried if standard pharmacological treatment is ineffective
  - o patients should be aware that they only provide short-term relief (2-10 weeks)
- if conservative methods fail then refer for consideration of joint replacement

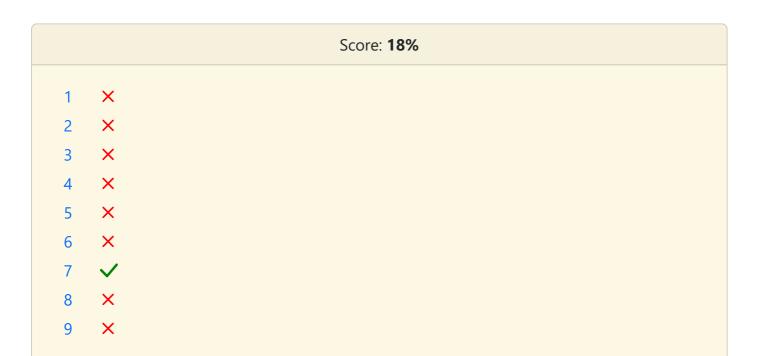


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High-yield textbook

### NICE 2022 osteoarthritis guidelines Suggest link Report broken link





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Ouestion 18 of 178





A 32-year-old man presents with a several-month history of recurrent ulcers. He experiences oral ulcers regularly, multiple at a time, which resolve after a week. He has also been experiencing genital ulcers. These are not painful. His past medical history includes two recent attendances to the eye casualty department; both times he was diagnosed as having uveitis.

Examination today is unremarkable except for a number of oral ulcers inside the mouth and some genital ulcers.

What would support the likely diagnosis?

Antinuclear antibodies	
Culture and serology of ulcers	
HLA-B27	
Pathergy test	
Rheumatoid factor	

Submit answer

Reference ranges ∨

## Score: **0%**1 2 3 4 5 6 7 8 9 -

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Question 18 of 178



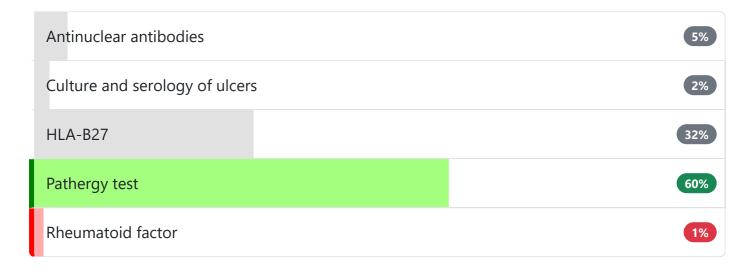
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A 32-year-old man presents with a several-month history of recurrent ulcers. He experiences oral ulcers regularly, multiple at a time, which resolve after a week. He has also been experiencing genital ulcers. These are not painful. His past medical history includes two recent attendances to the eye casualty department; both times he was diagnosed as having uveitis.

Examination today is unremarkable except for a number of oral ulcers inside the mouth and some genital ulcers.

What would support the likely diagnosis?



A positive pathergy test is suggestive of Behcet's syndrome

Important for me Less important



The history and examination here are suggestive of a diagnosis of Behcet's syndrome. This is a vasculitis that causes mucocutaneous, ophthalmological, vascular, gastrointestinal, and neurological manifestations. It is typically seen in patients aged 20-30 years, particularly in men. The classic presentation is that of oral and genital ulcers, with previous or current uveitis. There is no single test to accurately diagnose the condition; it is a clinical diagnosis. However, of the options listed, a **pathergy** test can support the diagnosis. This involves pricking the skin with a sterile needle, usually in the forearm. If the lesion becomes indurated within 48 hours, this is supportive of a diagnosis of Behcet's. The test is positive in approximately 60% of patients.

**Antinuclear antibodies** are not expected to be positive in Behcet's syndrome and so would not support the diagnosis. A positive result should prompt investigation of other autoimmune causes of the symptoms.

Culture and serology of the ulcers would not support a diagnosis of Behcet's. They would be

expected to be negative; any positive results should prompt reconsideration of the diagnosis.

**HLA-B27** would not be expected to be positive in Behcet's. A positive result would point towards seronegative spondyloarthropathies and would warrant further investigation.

**Rheumatoid factor** is not expected to be positive with Behcet's syndrome. If joint symptoms are also present, it can be useful to rule out rheumatoid arthritis but does not explicitly support the diagnosis of Behcet's.



Next question >

### Behcet's syndrome \*

Behcet's syndrome is a complex multisystem disorder associated with presumed autoimmunemediated inflammation of the arteries and veins. The precise aetiology has yet to be elucidated however. The classic triad of symptoms are oral ulcers, genital ulcers and anterior uveitis

### **Epidemiology**

- more common in the eastern Mediterranean (e.g. Turkey)
- more common in men (complicated gender distribution which varies according to country. Overall, Behcet's is considered to be more common and more severe in men)
- tends to affect young adults (e.g. 20 40 years old)
- associated with HLA B51
- around 30% of patients have a positive family history

### **Features**

- classically: 1) oral ulcers 2) genital ulcers 3) anterior uveitis
- thrombophlebitis and deep vein thrombosis
- arthritis
- neurological involvement (e.g. aseptic meningitis)
- GI: abdo pain, diarrhoea, colitis
- erythema nodosum

### Diagnosis

- no definitive test
- diagnosis based on clinical findings
- positive pathergy test is suggestive (puncture site following needle prick becomes inflamed with small pustule forming)

<sup>\*</sup>more specifically HLA B51, a split antigen of HLA B5

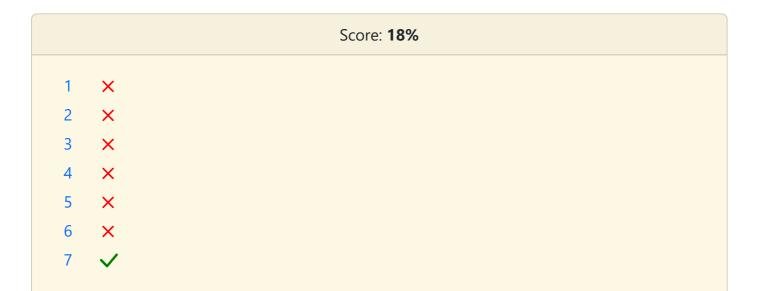


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Question 19 of 178





A 35-year-old man presents to his general practitioner with right elbow pain that has been present for 2 days and is exacerbated by movement. No other joints are affected and he denies stiffness. There is no past medical history of note and he is normally fit and well and plays sports regularly.

On examination, there is tenderness over the right elbow that is more prominent over the lateral aspect. There is no effusion or overlying skin changes.

What other examination finding best supports the likely diagnosis?

Pain on resisted finger abduction	
Pain on resisted forearm extension	
Pain on resisted forearm flexion	
Pain on resisted wrist extension	
Pain on resisted wrist flexion	

Submit answer

Reference ranges ∨

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Question 19 of 178



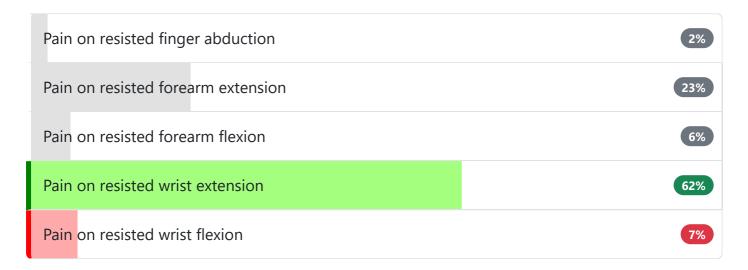
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A 35-year-old man presents to his general practitioner with right elbow pain that has been present for 2 days and is exacerbated by movement. No other joints are affected and he denies stiffness. There is no past medical history of note and he is normally fit and well and plays sports regularly.

On examination, there is tenderness over the right elbow that is more prominent over the lateral aspect. There is no effusion or overlying skin changes.

What other examination finding best supports the likely diagnosis?



Lateral epicondylitis: worse on resisted wrist extension/suppination whilst elbow extended

| Important for me | Less important |



This patient has lateral epicondylitis (tennis elbow) given the history of lateral elbow pain in a young sports player. The lateral epicondyle is an attachment point for the extensors of the wrist joint. Therefore, **pain on resisted wrist extension** is a diagnostic indicator.

**Pain on resisted finger abduction** tests the intrinsic hand muscles that would not be implicated in lateral epicondylitis.

**Pain on resisted forearm extension** tests the triceps muscles that attach from the scapula to the olecranon process of the elbow. Injury to the triceps muscle would not cause lateral epicondylitis.

**Pain on resisted forearm flexion** tests the biceps brachii and brachialis muscles that play no role in lateral epicondylitis.

Pain on resisted wrist flexion is a sign of medial epicondylitis (golfer's elbow) which is associated

with pain over the medial epicondyle secondary to a strain of the flexor compartment of the forearm.



Next question >

### Lateral epicondylitis \*

Lateral epicondylitis typically follows unaccustomed activity such as house painting or playing tennis ('tennis elbow'). It is most common in people aged 45-55 years and typically affects the dominant arm.

### **Features**

- pain and tenderness localised to the lateral epicondyle
- pain worse on wrist extension against resistance with the elbow extended or supination of the forearm with the elbow extended
- episodes typically last between 6 months and 2 years. Patients tend to have acute pain for 6-12 weeks

### Management options

- advice on avoiding muscle overload
- simple analgesia
- steroid injection
- physiotherapy



Next question >



### **Textbooks**

High-yield textbook

### Links

Clinical Knowledge Summaries

占 11 ♀ 6

Tennis elbow

Suggest link Report broken link

### Media



Understanding Tennis Elbow and Golfer's Elbow (Lateral & Medial Epicondylitis)

Rhesus Medicine - YouTube





Report broken media

Score: 18%

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### Question 20 of 178





A 32-year-old gentleman presented to his GP with an 8-week history of debilitating pain in his oral cavity and in his groin area. He had presented on numerous occasions to his GP with episodes of tiredness and non-specific malaise which each time was put down to non-specific viral illness. Eight months ago he was investigated by a gastroenterologist having presented with bloody diarrhoea and abdominal pain. He was diagnosed with a non-specific colitis of unknown origin which resolved spontaneously. He also suffered a solitary DVT of his left leg 6 years ago which was treated with oral anticoagulation. He smoked 20 cigarettes per day and consumed 20 units of alcohol per week. He was on no regular medication. Upon specific questioning, he denied joint pain or swelling. He also denied the presence of back pain. He was unaware of any family history as he was adopted from birth.

On examination, he appeared pale. His heart rate was 88 and blood pressure 118/78 mmHg. Examination of his cardiovascular system was unremarkable. Examination of his abdomen was likewise unremarkable. Examination of his oral mucosa revealed the presence of multiple aphthous ulceration. Examination of his external genitalia likewise revealed the presence of multiple shallow ulcers within his groin region. Examination of his joints was unremarkable.

Initial investigations revealed the following results:

Hb	139 g/l
Platelets	333 * 10 <sup>9</sup> /l
WBC	5.1 * 10 <sup>9</sup> /l
ESR	22 mm/hr
CRP	28 mg/l
Rheumatoid factor	negative
Anti CCP	negative
ANA	negative
HLA B27	positive

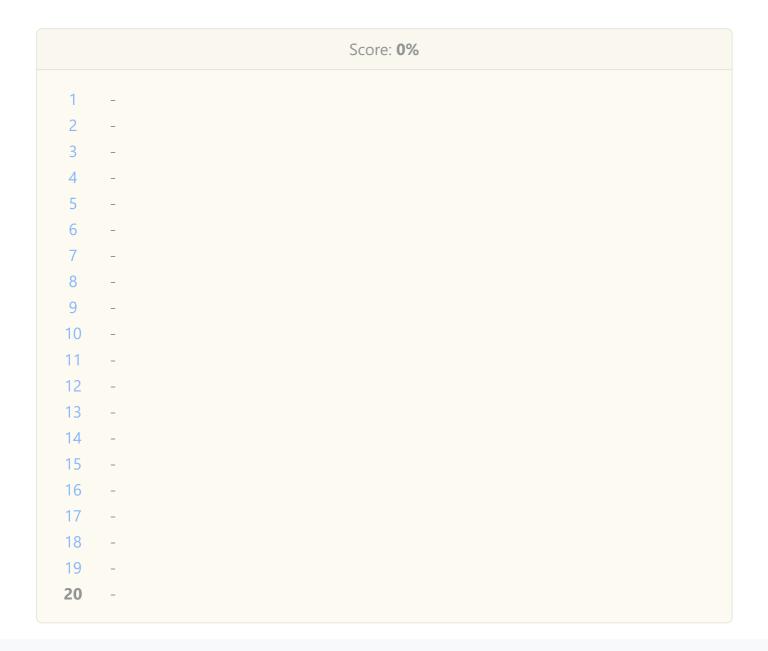
What is the most likely underlying diagnosis?

Seronegative arthritis	
Disseminated gonococcal infection	
Crohn's disease	

Coeliac disease	
Behcet's syndrome	

### Submit answer

Reference ranges ✓







Ouestion 20 of 178



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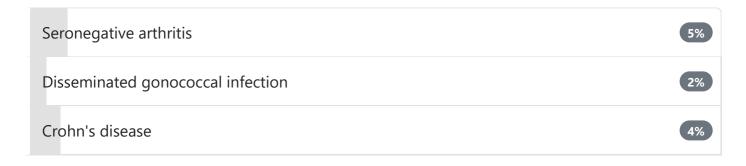
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ESR	22 mm/hr
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Anti CCP	negative
ANA	negative
HLA B27	positive

What is the most likely underlying diagnosis?

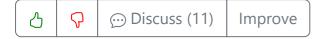


### Behcet's syndrome





This gentleman presents with a combination of malaise, oral and genital ulceration, colitis and iritis. Of the above options, Behcets syndrome is the only option that unifies this combination. Note that HLA B27 is positive in 10 % of the population in the absence of seronegative arthritis. Crohn's disease may present with a colitis and aphthous ulceration as well as iritis, but it would be difficult to account for the past deep vein thrombosis.



Next question >

### Behcet's syndrome \*

Behcet's syndrome is a complex multisystem disorder associated with presumed autoimmunemediated inflammation of the arteries and veins. The precise aetiology has yet to be elucidated however. The classic triad of symptoms are oral ulcers, genital ulcers and anterior uveitis

### **Epidemiology**

- more common in the eastern Mediterranean (e.g. Turkey)
- more common in men (complicated gender distribution which varies according to country. Overall, Behcet's is considered to be more common and more severe in men)
- tends to affect young adults (e.g. 20 40 years old)
- associated with HLA B51
- around 30% of patients have a positive family history

### Features

- classically: 1) oral ulcers 2) genital ulcers 3) anterior uveitis
- thrombophlebitis and deep vein thrombosis
- arthritis
- neurological involvement (e.g. aseptic meningitis)
- GI: abdo pain, diarrhoea, colitis
- erythema nodosum

### Diagnosis

• no definitive test

- diagnosis based on clinical findings
- positive pathergy test is suggestive (puncture site following needle prick becomes inflamed with small pustule forming)

\*more specifically HLA B51, a split antigen of HLA B5

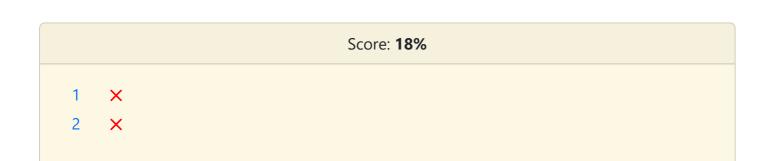


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### Question 21 of 178





A 54-year-old woman attends with shortness of breath and a non-productive cough. She has a past medical history of rheumatoid arthritis. Her drug history is as follows: methotrexate, folic acid, and omeprazole. Observations are as follows: heart rate 95 beats per minute, respiratory rate 20 breaths per minute, blood pressure 140/85 mmHg, temperature 37.1°C, and SpO2 94% on air.

### Blood results are as follows:

	Hb	125 g/L	Male: (135-180) Female: (115 - 160)
	Platelets	420 * 10 <sup>9</sup> /L	(150 - 400)
	WBC	14.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)

A high-resolution CT (HRCT) is performed:

HRCT Symmetrical and diffusely distributed ground-glass opacities with a basal predominance

### What treatment is indicated?

Co-amoxiclav and clarithromycin	
Co-trimoxazole	
Co-trimoxazole and prednisolone	
Prednisolone	
Prednisolone and folinic acid	

Submit answer

Reference ranges ∨

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Question 21 of 178



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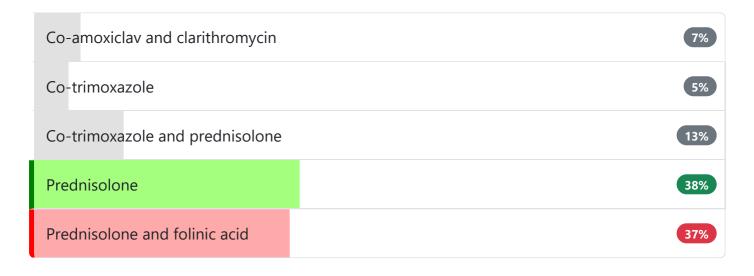
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WBC	14.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)

A high-resolution CT (HRCT) is performed:

HRCT Symmetrical and diffusely distributed ground-glass opacities with a basal predominance

What treatment is indicated?



Methotrexate may cause pneumonitis - typically presents with cough, dyspnoea and fever

| Important for me | Less important |

**Prednisolone** is correct. Steroids are the cornerstone of treatment for methotrexate pneumonitis.

**Co-amoxiclav and clarithromycin** is incorrect. This combination of antibiotics can be used to treat severe community-acquired pneumonia. The radiological appearances, in this case, are more suggestive of pneumonitis rather than pneumonia.

Co-trimoxazole is incorrect. This drug can be used to treat pneumocystis pneumonia (PCP) which

is a serious infection caused by the fungus *Pneumocystis jirovecii*. Although the patient is on low-dose methotrexate, this is not particularly immunosuppressive, making PCP a less likely diagnosis.

**Co-trimoxazole and prednisolone** is incorrect. The addition of steroids to co-trimoxazole can be used for severe cases of PCP characterised by a PaO2 < 8 kPa.

**Prednisolone and folinic acid** is incorrect. The clinical and radiological features are in keeping with methotrexate pneumonitis which should be managed with high-dose steroids. Indications for folinic acid include bone marrow toxicity which is not present in this case.



Next question >

### Methotrexate \*

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

### Indications

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - typically develops within a year of starting treatment, either acutely or subacutely
  - o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

## Prescribing methotrexate

- methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

## Interactions

- avoid prescribing <u>trimethoprim</u> or <u>co-trimoxazole</u> concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

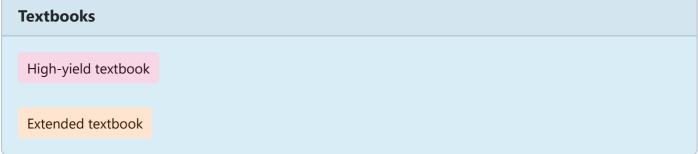
## Methotrexate toxicity

the treatment of choice is folinic acid

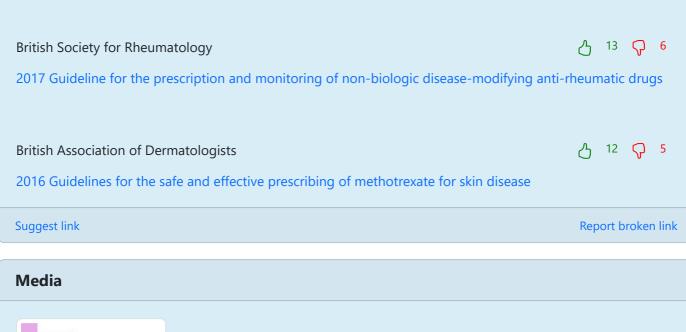


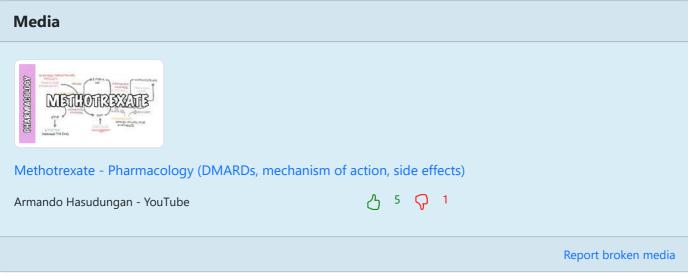
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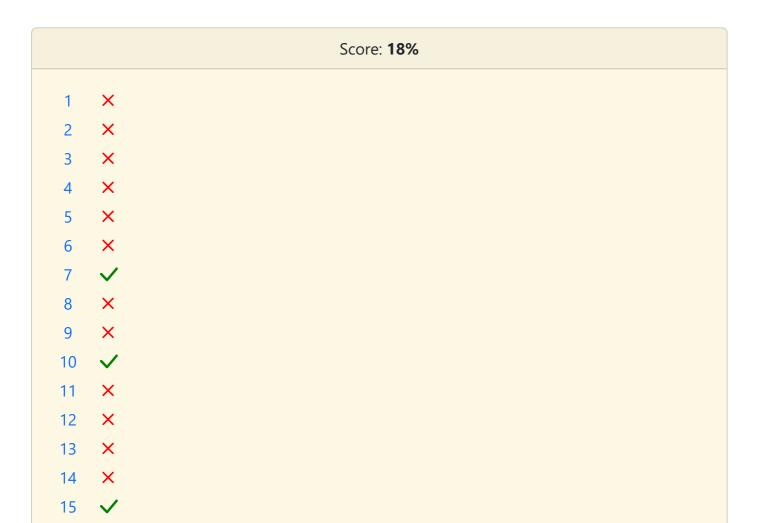












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## Question 22 of 178

 $\bowtie$ 



A 22-year-old woman presents to her general practitioner with a longstanding history of fatigue and generalised body aches. She was formerly an avid sportsperson, but over the years has become increasingly tired and has twisted her ankles on several occasions. Her past medical history includes recurrent dislocation of her right shoulder.

On examination, she has soft, elastic skin. She demonstrates joint hypermobility in multiple joints.

What ocular feature is also associated with this condition?

Angioid streaks	
Cataracts	
Hypermetropia	
Uveitis	
Yellow sclera	

Submit answer

Reference ranges ∨

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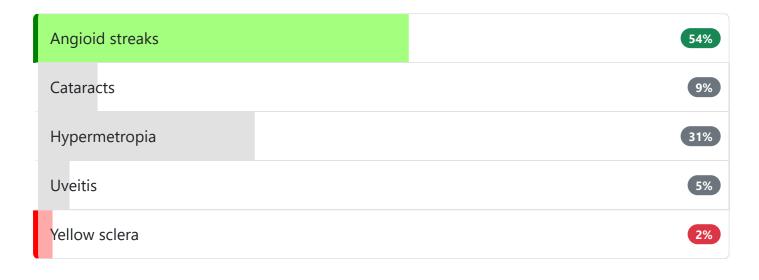




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On examination, she has soft, elastic skin. She demonstrates joint hypermobility in multiple joints.

What ocular feature is also associated with this condition?



Ehlers-Danlos syndrome: angioid retinal streaks is a feature

Important for me Less important



The history and examination are suggestive of Ehlers-Danlos syndrome (EDS), an inherited connective tissue disorder. Typical features include joint and skin hypermobility. Patients may have overlapping features of chronic fatigue syndrome and have a history of recurrent, easy dislocation of joints. Of the ocular features listed here, the only one with a clear association with EDS is **angioid streaks**. These appear as bilateral, narrow, irregular lines deep to the retina, appearing to radiate from the optic disc. These are usually asymptomatic but can lead to choroidal neovascularisation.

There is no clear association between **cataracts** and EDS. There are some conflicting reports on the incidence of cataracts in patients with EDS, but angioid retinal streaks have a much clearer association with the condition.

**Hypermetropia** is not associated with EDS. Rather, patients with EDS appear to have a higher prevalence of myopia than the general population.

**Uveitis** is not associated with EDS. It is linked to several autoimmune and rheumatological conditions.

Blue sclera, rather than **yellow sclera**, are associated with EDS. This is thought to be due to the loss of collagen in the eye.



Next question >

## **Ehler-Danlos syndrome**

Ehler-Danlos syndrome is an autosomal dominant connective tissue disorder that mostly affects type III collagen. This results in the tissue being more elastic than normal leading to joint hypermobility and increased elasticity of the skin.

Features and complications

- elastic, fragile skin
- joint hypermobility: recurrent joint dislocation
- easy bruising
- aortic regurgitation, mitral valve prolapse and aortic dissection
- subarachnoid haemorrhage
- angioid retinal streaks



Next question >





# Media Ehler-Danlos syndrome Armando Hasudungan - YouTube ♂ 1 ♀ 0 Report broken media

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The radiograph below was taken from a patient who presented with pain, swelling and erythema of the right knee.



## What is the diagnosis?

Osteoarthritis	
Rheumatoid arthritis	
Pseudogout	
Gout	
Tibial plateau fracture	

Submit answer

Reference ranges ∨

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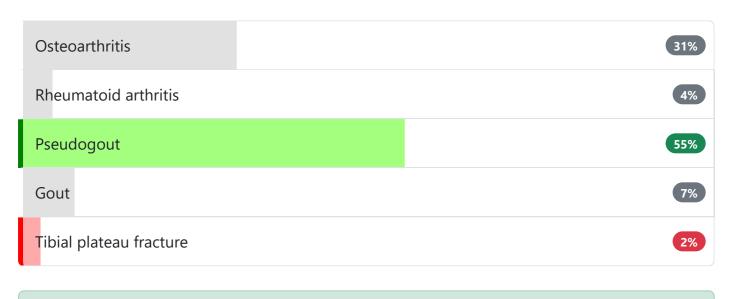
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The radiograph below was taken from a patient who presented with pain, swelling and erythema of the right knee.



## What is the diagnosis?



Chondrocalcinosis helps to distinguish pseudogout from gout

Important for me Less important



The radiograph demonstrates chondrocalcinosis (visible calcification of cartilage), a sign pathognomonic of pseudogout.



Next question >

## Pseudogout \*

Pseudogout is a form of microcrystal synovitis caused by the deposition of calcium pyrophosphate dihydrate crystals in the synovium. For this reason, it is now more correctly termed acute calcium pyrophosphate crystal deposition disease.

Pseudogout is strongly associated with increasing age. Patients who develop pseudogout at a younger age (e.g. < 60 years) usually have some underlying risk factor, such as:

- haemochromatosis
- hyperparathyroidism
- low magnesium, low phosphate
- acromegaly, Wilson's disease

## **Features**

- knee, wrist and shoulders most commonly affected
- joint aspiration: weakly-positively birefringent rhomboid-shaped crystals
- x-ray: chondrocalcinosis
  - o in the knee this can be seen as linear calcifications of the meniscus and articular cartilage

## Management

- aspiration of joint fluid, to exclude septic arthritis
- NSAIDs or intra-articular, intra-muscular or oral steroids as for gout



Next question >



## Textbooks High-yield textbook Extended textbook

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Question 24 of 178





A 34-year-old man with no fixed abode attends the emergency department with severe pain in his testicles and joint aches and pains. He has also noticed his left arm has been clumsy and that he has been dragging his left foot.

On examination, his scrotum is swollen and the right testicle is enlarged and extremely tender to touch. Triceps and brachioradialis reflexes are reduced, there is weakness in wrist extension and thumb abduction, with reduced sensation over the posterior forearm, and he has left-sided foot drop. He has purple, mesh-like discolouration of the skin on his lower limbs.

Chest x-ray: clear lung fields

What of the following is most in keeping with the likely diagnosis?

Eosinophilia	
Positive borrelia burgdorferi serology	
Positive cryoglobulins	
Positive hepatitis B serology	
Testicular biopsy showing non-caseating granulomas	

Submit answer

Reference ranges  $\vee$ 

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Question 24 of 178



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Chest x-ray: clear lung fields

What of the following is most in keeping with the likely diagnosis?

Eosinophilia	5%
Positive borrelia burgdorferi serology	6%
Positive cryoglobulins	25%
Positive hepatitis B serology	56%
Testicular biopsy showing non-caseating granulomas	8%

Systemic vasculitic symptoms in the presence of hepatitis B signs and in the absence of pulmonary symptoms/signs suggests a diagnosis polyarteritis nodosa

Important for me Less important



The patient has orchitis, arthralgia, livedo reticularis, and mononeuritis multiplex. The most likely diagnosis is polyarteritis nodosa. This is strongly associated with hepatitis B, which is more commonly seen in people with no fixed abode.

**Positive hepatitis B serology** is correct. 30% of patients with polyarteritis nodosa have positive hepatitis B serology.

**Testicular biopsy showing non-caseating granuloma** is incorrect. Polyarteritis nodosa is a necrotising vasculitis which does not lead to granuloma formation. Non-caseating granulomas may be seen in other inflammatory conditions such as sarcoidosis and Crohn's. The symptoms

here are not in keeping with a diagnosis of sarcoidosis, there is no lymphadenopathy, no respiratory symptoms, and a chest x-ray has been reported as normal.

**Positive borrelia burgdorferi serology** is incorrect. Lyme disease may be a differential diagnosis, and it would be reasonable to test for it if there was diagnostic uncertainty, but there is no history of tick exposure and there is no characteristic erythema migrans rash.

**Eosinophilia** is incorrect. Eosinophilia is associated with Eosinophilic granulomatosis with polyangiitis. Symptoms usually include respiratory and nasal problems, but it can be associated with peripheral neuropathy and arthralgia as seen here. It would be extremely unusual for EGPA to cause orchitis, and so it is not the most likely diagnosis here.

**Positive cryoglobulins** is incorrect. Cryoglobulinaemia is a condition where cryoglobulin proteins clump at lower temperatures, this is usually in the extremities such as fingers and toes. There is usually purpura (which is not present in this case), joint pain, hyperviscosity symptoms, and weakness. Sensory abnormalities are not typical. When it presents with cryoglobulinaemic vasculitis, it is a cutaneous small-vessel vasculitis and so commonly presents with infarctions and necrosis of tissues such as the skin, this is not a feature described in the stem.



Next question >

## Polyarteritis nodosa 🖈

Polyarteritis nodosa (PAN) is a vasculitis affecting medium-sized arteries with necrotizing inflammation leading to aneurysm formation. PAN is more common in middle-aged men and is associated with hepatitis B infection.

### **Features**

- fever, malaise, arthralgia
- weight loss
- hypertension
- mononeuritis multiplex, sensorimotor polyneuropathy
- testicular pain
- livedo reticularis
- haematuria, renal failure
- perinuclear-antineutrophil cytoplasmic antibodies (ANCA) are found in around 20% of patients with 'classic' PAN
- hepatitis B serology positive in 30% of patients



© Image used on license from DermNet NZ

## Livedo reticularis



© Image used on license from Radiopaedia

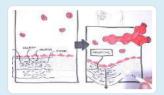
Angiogram from a patient with polyarteritis nodosa. Both kidneys demonstrate beading and numerous microaneurysms affecting the intrarenal vessels. Similar changes are seen affecting the intrahepatic vessels with a few small microaneurysms noted. The proximal branches of the SMA appears normal; however there are no normal straight arteries from the jejunal arteries and lack of normal anastomotic arcades and loops. This is associated with multiple microaneurysms.



## Textbooks High-yield textbook

Extended textbook

## Media



Polyarteritis Nodosa and Kawasaki Disease (Medium Vessel Vasculitis) - Symptoms, pathophysiology

Armando Hasudungan - YouTube



Report broken media

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Question 25 of 178





A 75-year-old woman presents to the emergency department with a painful swollen wrist. There is no history of trauma. She has recently completed antibiotic treatment for a urinary tract infection. She has a background of gastro-oesophageal reflux disease and is on long-term omeprazole.

## Observations:

- Heart rate 88 beats per minute
- Blood pressure 132/77 mmHg
- Respiratory rate 18/minute
- Oxygen saturation 97% on room air
- Temperature 37.3°C

On examination, there is a warm, tender swollen left wrist with a restricted range of motion.

## Blood tests:

Hb	121 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10 <sup>9</sup> /L	(150 - 400)
WBC	8.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	138 mmol/L	(135 - 145)
K <sup>+</sup>	4.2 mmol/L	(3.5 - 5.0)
Urea	8.9 mmol/L	(2.0 - 7.0)
Creatinine	124 µmol/L	(55 - 120)
CRP	41 mg/L	(< 5)
Magnesium	0.54 mmol/L	(0.7-1.00)

What is most likely to be found on a joint aspirate in this case?

Escherichia coli	
Methicillin-resistant Staphylococcus aureus (MRSA)	
Staphylococcus aureus	
Negatively birefringent needle-shaped crystals	

## Submit answer

Reference ranges  $\checkmark$ 

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Question 25 of 178



 $\Box$ 



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Creatinine	124 µmol/L	(55 - 120)
CRP	41 mg/L	(< 5)
Magnesium	0.54 mmol/L	(0.7-1.00)

What is most likely to be found on a joint aspirate in this case?

Escherichia coli	9%
Methicillin-resistant Staphylococcus aureus (MRSA)	1%
Staphylococcus aureus	15%
Negatively birefringent needle-shaped crystals	28%

Pseudogout - weakly positively birefringent rhomboid-shaped crystals

Important for me Less important

**Positively birefringent rhomboid-shaped crystals** is correct. This elderly woman presents with monoarthritis of the wrist in close proximity to an infective episode. The most common explanation for this is an episode of pseudogout. Hypomagnesemia (likely caused by omeprazole use) is a risk factor for this condition. An infection should be excluded but is not the most likely explanation. It is possible to have septic arthritis with a normal WCC and temperature. However, the absence of neutrophilia and significant pyrexia does point away from the diagnosis. Pseudogout is characterized by positively birefringent rhomboid-shaped crystals in the synovial fluid.

**Methicillin-resistant Staphylococcus aureus (MRSA)** is incorrect. This can cause septic arthritis but is an unlikely community-acquired cause of septic arthritis. The CRP may be elevated in both. It would be more likely to occur in a hospital or after an intervention/procedure.

**Escherichia coli** is incorrect. Intercurrent urinary tract infections increase the probability that septic arthritis may be caused by a gram-negative rather than a gram-positive organism. This is still a rarer event than a flare of pseudogout, however.

**Staphylococcus aureus** is incorrect. This is the most common organism causing septic arthritis. However, as previously mentioned, a more common cause of monoarthritis of the wrist in this age group, in this context, is pseudogout.

**Negatively birefringent needle-shaped crystals** is incorrect. This finding is typical of the monosodium urate crystals of gout. A monoarthritis of the wrist is more likely to be pseudogout than gout.



Next question >

## Pseudogout \*

Pseudogout is a form of microcrystal synovitis caused by the deposition of calcium pyrophosphate dihydrate crystals in the synovium. For this reason, it is now more correctly termed acute calcium pyrophosphate crystal deposition disease.

Pseudogout is strongly associated with increasing age. Patients who develop pseudogout at a younger age (e.g. < 60 years) usually have some underlying risk factor, such as:

- haemochromatosis
- hyperparathyroidism
- low magnesium, low phosphate
- acromegaly, Wilson's disease

## **Features**

- knee, wrist and shoulders most commonly affected
- joint aspiration: weakly-positively birefringent rhomboid-shaped crystals
- x-ray: chondrocalcinosis
  - o in the knee this can be seen as linear calcifications of the meniscus and articular cartilage

## Management

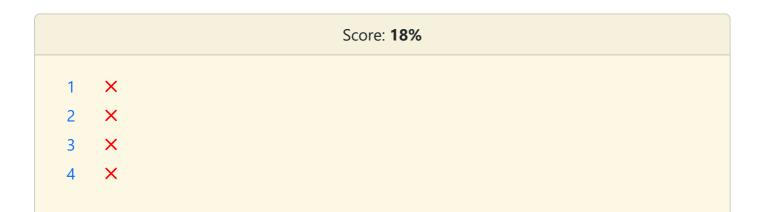
- aspiration of joint fluid, to exclude septic arthritis
- NSAIDs or intra-articular, intra-muscular or oral steroids as for gout



Next question >







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Question 26 of 178





A 34-year-old patient presents to her general practitioner complaining of a rash in both ears. She has no past medical history. She does not take any regular medications. She frequently goes on holiday to hot-weather climates.

On examination, there are red plaques with adherent scales in the concha of both ears.

A skin biopsy demonstrates an interface dermatitis.

Given the likely diagnosis, what is the appropriate treatment?

Intravenous belimumab	
Oral hydroxychloroquine	
Oral steroids	
Topical imiquimod	
Topical steroids	

Submit answer

Reference ranges ✓

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Score: 0%

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Question 26 of 178



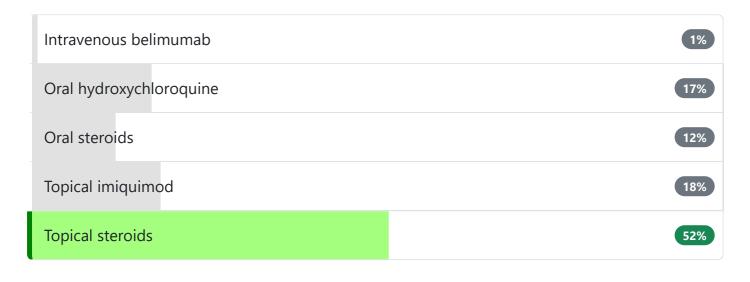


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A skin biopsy demonstrates an interface dermatitis.

Given the likely diagnosis, what is the appropriate treatment?



Discoid lupus erythematous - topical steroids → oral hydroxychloroquine

Important for me Less important



**Topical steroids** is the right answer. The patient presents with an erythematous rash with an adherent scale in both ears. This is a classic location for discoid lupus erythematosus (DLE) and is known as 'Schuster's sign' when it affects the conchal bowl of both ears. A biopsy demonstrating interface dermatitis is typical. The first-line treatment of discoid lupus is topical steroids.

**Intravenous belimumab** is incorrect. This is indicated as add-on therapy for those patients with systemic lupus erythematosus with severe disease not responding to conventional therapy. It is particularly indicated for those with active musculoskeletal and cutaneous (which may involve DLE) disease. It is not indicated for isolated DLE as a first-line therapy.

**Oral hydroxychloroquine** is incorrect. Topical steroids should be tried in the first instance. If this strategy is ineffective then oral hydroxychloroquine can be commenced. It should be noted that if discoid lupus was a manifestation of systemic lupus erythematosus then hydroxychloroquine should be commenced. Discoid lupus erythematosus can exist as an isolated skin disease with a

negative antinuclear antibody (i.e. not as part of SLE) and in this setting, topical steroids alone are a reasonable first option.

**Oral steroids** is incorrect. Oral corticosteroids are associated with significant toxicity with long-term use and therefore are reserved only for severe or refractory cases.

**Topical imiquimod** is incorrect. This is an immunomodulator that is used in the treatment of genital warts, superficial basal cell carcinomas, and actinic keratoses. The description of this bilateral auricular conchal rash is not typical of a basal cell carcinoma (BCC), which is usually described as a lesion having pearly rolled edges. Actinic (solar) keratoses can cause scaly red plaques although they are more typically yellow. They tend to occur as a consequence of sun damage over time and 34 would be quite a young age to develop this type of lesion.



Next question >

## Discoid lupus erythematosus \*

Discoid lupus erythematosus is a benign disorder generally seen in younger females. It very rarely progresses to systemic lupus erythematosus (in less than 5% of cases). Discoid lupus erythematosus is characterised by follicular keratin plugs and is thought to be autoimmune in aetiology

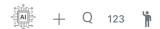
## **Features**

- erythematous, raised rash, sometimes scaly
- may be photosensitive
- more common on face, neck, ears and scalp
- lesions heal with atrophy, scarring (may cause scarring alopecia), and pigmentation

## Management

- topical steroid cream
- oral antimalarials may be used second-line e.g. hydroxychloroquine
- avoid sun exposure





Next question >







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A 47-year-old lady presents to her General Practitioner complaining of episodes of joint swelling, morning stiffness, weight loss, myalgia and general malaise. She is wondering if she has hyperthyroidism, as both her grandmother and her mother required thyroidectomy when they were younger.

Examination reveals a temperature of 37.1°C and a mild erythema around the nose, but sparing the nasolabial folds. There is no proximal myalgia and cardiovascular and respiratory examination is unremarkable. There is no goitre.

Blood investigations come back and the raised ESR prompts performance of an autoimmune profile:

Rheumatoid Factor	Positive
Anti-CCP	Negative
ANA	1:320
ANCA	Negative
Anti-DsDNA	Positive
C3/C4 levels	Normal
Anti-RNP	Positive

TSH	4.5 miU/I
T4	12 pmol/l

Given the above results what is the most likely diagnosis?

Palindromic Rheumatoid Arthritis	
Polymyalgia rheumatica	
Systemic lupus erythematosus	
Hyperthyroidism	
Mixed Connective Tissue Disorder	

Submit answer

		Score: 0%
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Question 27 of 178



 $\Box$ 



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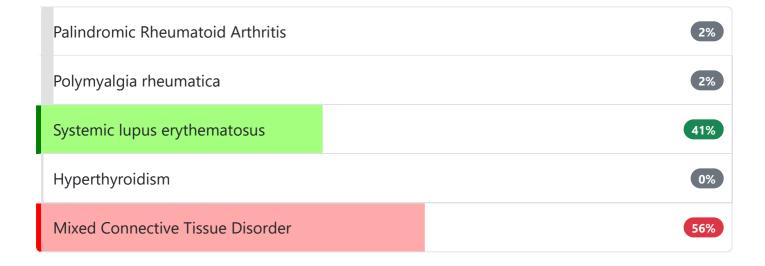
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ANCA	Negative
Anti-DsDNA	Positive
C3/C4 levels	Normal
Anti-RNP	Positive

TSH	4.5 miU/l
T4	12 pmol/l

Given the above results what is the most likely diagnosis?



Anti-RNP antibodies can be positive in systemic lupus erythematosus in nearly 40% of patients

Important for me Less important

This lady has an array of positive antibodies associated with rheumatological conditions. It is important however here to pay attention to the actual symptomatology displayed by the patient and her presenting complaints. The gender, age and history lean towards the diagnosis of a systemic autoimmune condition, with Systemic lupus erythematosus appearing as the most plausible explanation, given her examination findings. The fleeting joint swelling is common in SLE, alongside the non-specific symptoms and the characteristic butterfly rash. The possibility of palindromic rheumatoid arthritis could explain this fleeting arthralgia but does not account for the rest of the signs observed. Polymyalgia rheumatica would be rare in the patient's age and would not exhibit the findings seen. Mixed Connective Tissue Disorder possibility is raised by the presence of anti-RNP antibodies, but one should bear in mind the absence of other overlap features and that anti-RNP antibodies can be found in up to 40% of patients with SLE. Finally Hyperthyroidism is purely a red herring, with patients commonly presenting in primary care with a fixed belief based on prior experiences, whilst actually the pathological process is different.



Next question >

# Systemic lupus erythematosus: investigations \*

#### **Antibodies**

- 99% are ANA positive
  - o this high sensitivity makes it a useful rule out test, but it has low specificity
- 20% are rheumatoid factor positive
- anti-dsDNA: highly specific (> 99%), but less sensitive (70%)
- anti-Smith: highly specific (> 99%), sensitivity (30%)
- also: anti-U1 RNP, SS-A (anti-Ro) and SS-B (anti-La)

#### Monitoring

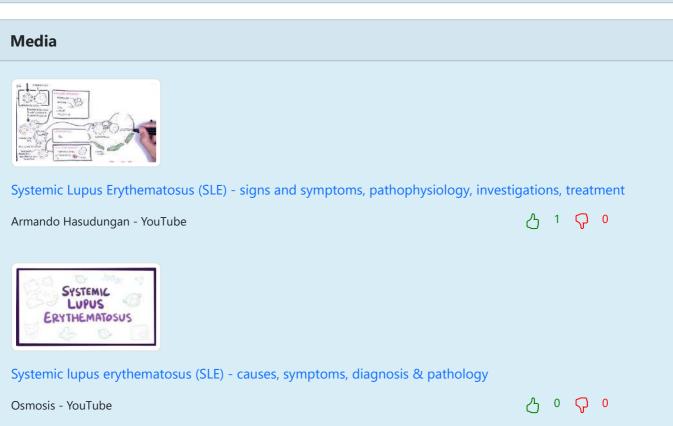
- inflammatory markers
  - ESR is generally used
  - during active disease the CRP may be normal a raised CRP may indicate underlying infection
- complement levels (C3, C4) are low during active disease (formation of complexes leads to consumption of complement)
- anti-dsDNA titres can be used for disease monitoring (but note not present in all patients)

Next question >









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#### Question 28 of 178





A 76-year-old lady is seen in rheumatology outpatient clinic, as she was previously diagnosed with Giant Cell Arteritis 4 years ago. Unfortunately since then she has remained on prednisolone 8 mg, and seems to be unable to go on a lower dose; previous attempts (x3) have resulted in excruciating temporal headaches, weakness and fatigue. The most recent attempt was accompanied with mild visual loss, which required her hospitalisation and caused her significant distress.

She is also on zoledronic acid for osteoporosis, as her most recent DEXA scan indicated a T-score on the neck of femur of -2.8. A recent spinal x-ray also revealed a number of vertebral fractures.

She comes wondering if steroid will continue to be her treatment, as she is quite concerned about their side effects, including diabetes (she tells you her sister recently lost 2 toes from diabetes) and weight gain.

What options exist in the management of this lady's giant cell arteritis?

Methotrexate	
Continue with current dose of prednisolone	
Attempt a reduction in prednisolone dosage	
Etanercept	
Ciclosporin	

Submit answer

Reference ranges ✓

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Question 28 of 178



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She comes wondering if steroid will continue to be her treatment, as she is quite concerned about their side effects, including diabetes (she tells you her sister recently lost 2 toes from diabetes) and weight gain.

What options exist in the management of this lady's giant cell arteritis?

Methotrexate					31%
Continue with c	urrent dose	of prednisolone			18%
Attempt a reduction in prednisolone dosage				6%	
Etanercept					28%
Ciclosporin					18%

Methotrexate can be used as a steroid-sparing agent in difficult to control, frequently relapsing giant cell arteritis

Important for me Less important



This lady has Giant Cell Arteritis that is difficult to manage, ridden with numerous relapses. Even though it is commonly accepted that low dose steroids can continue in elderly patients provided that no side effects have arisen, this is a lady with osteoporosis, who is also having family history of type II diabetes mellitus and is concerned herself about the long-time use of steroid. As per British Society of Rheumatology guidelines an appropriate alternative would include methotrexate, provided of course that she has normal liver function and all her pre-methotrexate testing is normal. Etanercept and ciclosporin, although used in the past in the treatment of difficult to

manage GCA, studies have no shown any added benefit from the use of etanercept or ciclosporin in the management of such cases of GCA.



Next question >

# Temporal arteritis \*

Temporal arteritis (also known as giant cell arteritis: GCA) is a vasculitis of unknown cause that affects medium and large-sized vessels arteries. It occurs in those over 50 years old, with a peak incidence in patients who are in their 70s.

It requires early recognition and treatment to minimize the risk of complications such as permanent loss of vision. Hence, when temporal arteritis is suspected, treatment must be started promptly with high-dose prednisolone as well as urgent referral for assessment by a specialist.

There is an overlap between temporal arteritis and polymyalgia rheumatica (PMR) - around 50% of patients will have features of PMR.

#### **Features**

- typically patient > 60 years old
- usually rapid onset (e.g. < 1 month)</li>
- headache (found in 85%)
- jaw claudication (65%)
- vision testing is a key investigation in all patients
  - anterior ischemic optic neuropathy accounts for the majority of ocular complications. It results from occlusion of the posterior ciliary artery (a branch of the ophthalmic artery) → ischaemia of the optic nerve head. Fundoscopy typically shows a swollen pale disc and blurred margins
  - may result in temporary visual loss amaurosis fugax
  - permanent visual loss is the most feared complication of temporal arteritis and may develop suddenly
  - diplopia may also result from the involvement of any part of the oculomotor system (e.g. cranial nerves)
- tender, palpable temporal artery
- around 50% have features of PMR: aching, morning stiffness in proximal limb muscles (not weakness)
- also lethargy, depression, low-grade fever, anorexia, night sweats

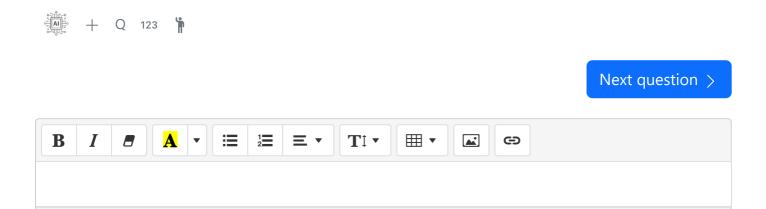
#### Investigations

- raised inflammatory markers
  - ESR > 50 mm/hr (note ESR < 30 in 10% of patients)</li>

- o CRP may also be elevated
- temporal artery biopsy
  - o skip lesions may be present
- note creatine kinase and EMG normal

#### **Treatment**

- urgent high-dose glucocorticoids should be given as soon as the diagnosis is suspected and before the temporal artery biopsy
  - o if there is no visual loss then high-dose prednisolone is used
  - if there is evolving visual loss IV methylprednisolone is usually given prior to starting high-dose prednisolone
  - o there should be a dramatic response, if not the diagnosis should be reconsidered
- urgent ophthalmology review
  - o patients with visual symptoms should be seen the same-day by an ophthalmologist
  - visual damage is often irreversible
- other treatments
  - bone protection with bisphosphonates is required as long, tapering course of steroids is required
  - o low-dose aspirin is sometimes given to patients as well, although the evidence base supporting this is weak









2010 BSR and BHPR guidelines for the management of giant cell arteritis

Suggest link Report broken link

#### Media



**Temporal Arteritis** 

Pixorize - YouTube







Temporal arteritis

Khan Academy - YouTube







Temporal artery biopsy

Oculoplastics.info - YouTube







Giant cell Arteritis and Takayasu arteritis (Large Vessel Vasculitis) - signs, pathophysiology

Armando Hasudungan - YouTube





Report broken media

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Question 29 of 178





A 38-year-old lady presents to her General Practitioner worried regarding a crusty patch of skin on her nose. The doctor proceeds to examine her and identifies a pearly nodule with shiny borders that is roughly 0.3 cm x 0.3 cm.

She is known to suffer from inflammatory bowel disease and is currently on Azathioprine. Despite advice from her gastroenterologist she is still smoking.

What risk factor in her history can be identified related to her presentation?

Female gender	
Age above 30 years old	
Inflammatory bowel disease	
Smoking history	
Azathioprine use	

Submit answer

Reference ranges ∨

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Question 29 of 178



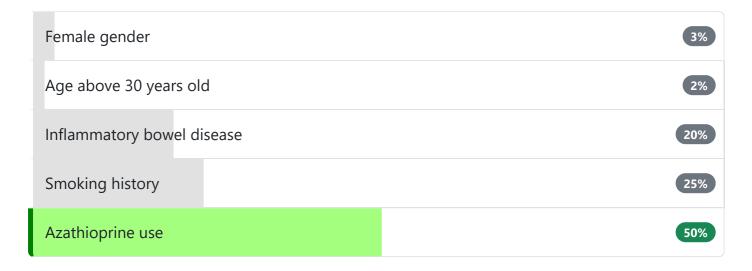
 $\Box$ 



A 38-year-old lady presents to her General Practitioner worried regarding a crusty patch of skin on her nose. The doctor proceeds to examine her and identifies a pearly nodule with shiny borders that is roughly 0.3 cm x 0.3 cm.

She is known to suffer from inflammatory bowel disease and is currently on Azathioprine. Despite advice from her gastroenterologist she is still smoking.

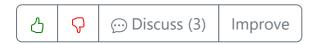
What risk factor in her history can be identified related to her presentation?



Azathioprine is associated with increased risk of non-melanoma skin cancer

| Important for me | Less important |

The lesion described is that of basal cell carcinoma. The only risk factor associated with this is azathioprine.



Next question >

# Azathioprine \*

Azathioprine is metabolised to the active compound mercaptopurine, a purine analogue that inhibits purine synthesis. A thiopurine methyltransferase (TPMT) test may be needed to look for individuals prone to azathioprine toxicity.

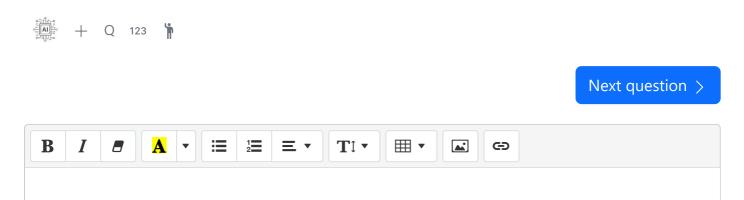
Adverse effects include

bone marrow depression

- o consider a full blood count if infection/bleeding occurs
- nausea/vomiting
- pancreatitis
- increased risk of non-melanoma skin cancer

A significant interaction may occur with <u>allopurinol</u> and hence lower doses of azathioprine should be used.

Azathioprine is generally considered safe to use in pregnancy.







Score: 18%

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Question 30 of 178





A 78-year-old man has a cervical spine film after falling down the stairs at his house. He has no history of musculoskeletal problems, including no neck or arm pain.

The cervical spine film is shown below:





What does the cervical spine film show?

Diffuse idiopathic skeletal hyperostosis	
Multiple myeloma	
Cervical rib	

Spondylosis of the cervical spine	
Ankylosing spondylitis	

## Submit answer

Reference ranges ✓

		Score: 0%
1	-	
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Question 30 of 178

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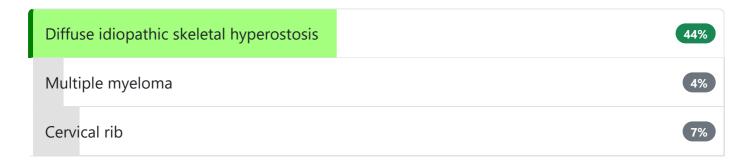
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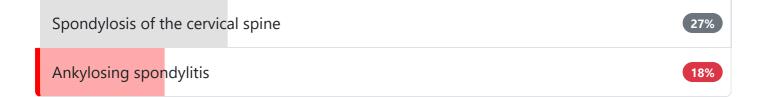
A 78-year-old man has a cervical spine film after falling down the stairs at his house. He has no history of musculoskeletal problems, including no neck or arm pain.

The cervical spine film is shown below:



What does the cervical spine film show?





The cervical film shows ossification of the anterior longitudinal ligament only sparing the segments C2/3.



Next question >

# Diffuse idiopathic skeletal hyperostosis \*

Diffuse idiopathic skeletal hyperostosis (DISH) describes the relatively common finding of ossification at sites of tendinous and ligamentous insertion of the spine. It tends to be seen in elderly patients.

DISH is generally asymptomatic.



© Image used on license from Radionaedia

0

Ossification of the anterior longitudinal ligament only sparing the segments C2/3 consistent with DISH



Next question >



#### **Textbooks**

High-yield textbook

# 

Score: **18%** × 1 2 × X 3 X 4 X 5 X 6 7 X 8 X 9 10 X 11 X 12 X 13 X 14 15 X 16 X 17 18 × X 19 20 X 21 X 22 23 X × 24 25

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Question 31 of 178





A 31-year-old woman has been referred to your clinic with multiple symptoms. She has reduced sensation in the 4th and 5th digits, extreme fatigue, pain in her hands, and weight loss.

On examination, she has active synovitis in multiple metacarpophalangeal joints and reduced sensation over the 4th and 5th digits of the right hand. She also has a rash over her chest and back with large, ring-shaped lesions that overlap. Her chest is clear and her heart sounds are normal.

Pre-clinic blood tests sent in the community are as below:

Hb	109 g/L	Male: (135-180) Female: (115 - 160)
Platelets	434 * 10 <sup>9</sup> /L	(150 - 400)
WBC	7.4 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Neuts	6.0 * 10 <sup>9</sup> /L	(2.0 - 7.0)
Lymphs	0.3 * 10 <sup>9</sup> /L	(1.0 - 3.5)
Mono	0.7 * 10 <sup>9</sup> /L	(0.2 - 0.8)
Eosin	0.4 * 10 <sup>9</sup> /L	(0.0 - 0.4)
Na <sup>+</sup>	135 mmol/L	(135 - 145)
K <sup>+</sup>	3.6 mmol/L	(3.5 - 5.0)
Urea	3.5 mmol/L	(2.0 - 7.0)
Creatinine	60 µmol/L	(55 - 120)
CRP	43 mg/L	(< 5)

You send some more blood tests and the results are awaited.

Given the likely diagnosis, what would be the most appropriate treatment of choice for long-term management?

Azathioprine	
Cyclophosphamide	×
Hydroxychloroquine	×
Methotrexate	×

# Submit answer

Reference ranges  $\checkmark$ 

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Question 31 of 178



 $\Box$ 



A 31-year-old woman has been referred to your clinic with multiple symptoms. She has reduced sensation in the 4th and 5th digits, extreme fatigue, pain in her hands, and weight loss.

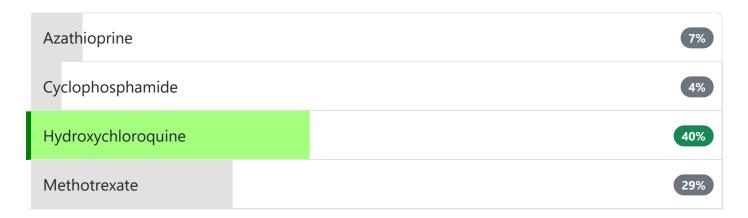
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WBC	7.4 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Neuts	6.0 * 10 <sup>9</sup> /L	(2.0 - 7.0)
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Urea	3.5 mmol/L	(2.0 - 7.0)
Creatinine	60 µmol/L	(55 - 120)
CRP	43 mg/L	(< 5)

You send some more blood tests and the results are awaited.

Given the likely diagnosis, what would be the most appropriate treatment of choice for long-term management?



## Hydroxychloroquine is the treatment of choice for SLE

Important for me Less important



The most likely diagnosis here is systemic lupus erythematosus. Known as the 'great mimic', SLE can present with a vast variety of symptoms. In this case, there is a description of a classic subacute cutaneous lupus rash, joint involvement, peripheral neuropathy, and extreme fatigue. The blood tests show anaemia, thrombophilia, and lymphopenia, all signs of active inflammatory disease. You would also expect a positive ANA.

**Hydroxychloroquine** is the correct answer. This is the treatment of choice for SLE.

**Azathioprine** is incorrect. Although this can be used in the management of SLE, it would not be the typical first-choice option. It may be considered if hydroxychloroquine is insufficient in managing non-renal manifestations of lupus after TPMT testing.

**Cyclophosphamide** is incorrect. This would not be the typical first-line option for long-term management of lupus particularly in this patient who is of childbearing age. It is not typically used long-term due to toxicity. In severe cases of lupus, cyclophosphamide is considered to gain rapid disease control.

**Methotrexate** is incorrect. Although this can be used in the management of SLE, it would not be the typical first-choice option. It may be considered if hydroxychloroquine is insufficient in managing non-renal manifestations of lupus.

**Prednisolone with lansoprazole cover** is incorrect. Prednisolone is associated with many complications including gastric ulceration, osteoporosis, and steroid-induced diabetes amongst others. It would not be an appropriate first-line long-term management strategy.



Next question >

# Systemic lupus erythematosus: management \*

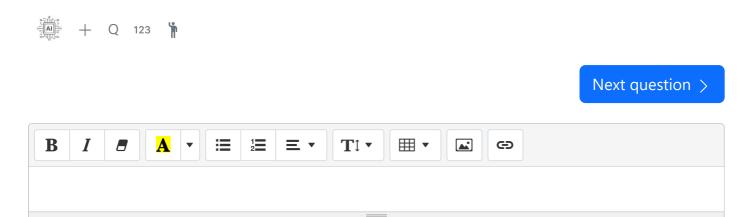
#### **Basics**

- NSAIDs
- sun-block

# Hydroxychloroquine

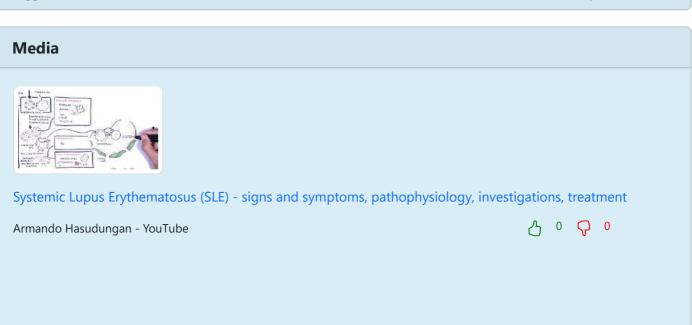
• the treatment of choice for SLE

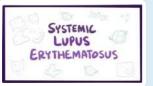
If internal organ involvement e.g. renal, neuro, eye then consider prednisolone, cyclophosphamide











Systemic lupus erythematosus (SLE) - causes, symptoms, diagnosis & pathology

Osmosis - YouTube





Report broken media

# Score: **18%** 1 X 2 X X 3 X 4 × 5 X 6 7 8 X X 9 10 X 11 X 12 13 X × 14 15 X 16 17 X 18 X 19 X 20 21 X X 22 23 X 24 X 25 26

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Question 32 of 178





A 25-year-old Greek man attends the emergency department with a fever. He states that this has been occurring most evenings for the past two weeks. At the onset of the fever, he also develops a rash and pain in his elbows, knees and ankles.

His observations are as follows: heart rate 110 beats per minute, blood pressure 115/70 mmHg, respiratory rate 16 breaths per minute, temperature 39.2°C, and SpO2 98% (on air). On examination, you note a maculopapular rash over his back and upper limbs. There is also pain and swelling of his wrists, knees and ankles. There is no evidence of lymphadenopathy or hepatosplenomegaly.

#### Blood results are as follows:

Hb	145 g/L	Male: (135-180) Female: (115 - 160)
Platelets	580 * 10 <sup>9</sup> /L	(150 - 400)
WBC	14.8 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	138 mmol/L	(135 - 145)
K <sup>+</sup>	3.8 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	68 µmol/L	(55 - 120)
CRP	68 mg/L	(< 5)
Ferritin	>20,000 µg/L	(30 - 400)

Bilirubin	10 μmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	24 u/L	(3 - 40)
γGT	42 u/L	(8 - 60)
Albumin	38 g/L	(35 - 50)

# What is the most likely diagnosis?

Adult-onset Still's Disease	
Familial Mediterranean fever (FMF)	
Hemophagocytic lymphohistiocytosis (HLH)	

Psoriatic arthritis	
Rheumatoid arthritis	

# Submit answer

Reference ranges ✓

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Question 32 of 178



 $\Box$ 



A 25-year-old Greek man attends the emergency department with a fever. He states that this has been occurring most evenings for the past two weeks. At the onset of the fever, he also develops a rash and pain in his elbows, knees and ankles.

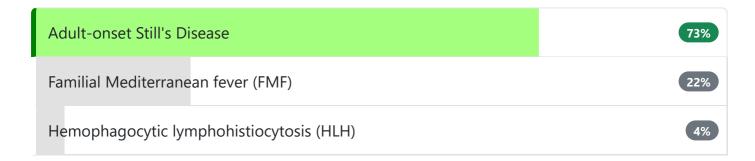
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CRP	68 mg/L	(< 5)
Ferritin	>20,000 µg/L	(30 - 400)

Bilirubin	10 μmol/L	(3 - 17)
ALP	88 u/L	(30 - 100)
ALT	24 u/L	(3 - 40)
γGT	42 u/L	(8 - 60)
Albumin	38 g/L	(35 - 50)

What is the most likely diagnosis?



Pyrexia in Still's disease has a characteristic pattern. It typically rises in the late afternoon/early evening in a daily pattern and accompanies a worsening of joint symptoms and rash

Important for me Less important



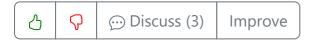
**Adult-onset Still's Disease** is correct. This condition can be extremely difficult to diagnose due to its rarity and clinical features which overlap with many other conditions. However, there are some clues which can point to this diagnosis including the typical daily fever which occurs in the late afternoon/early evening and accompanies a worsening of joint symptoms and 'salmon-pink' maculopapular rash. An extreme hyperferritinemia >20,000 µg/L is a further clue.

**Familial Mediterranean fever (FMF)** is incorrect. Familial Mediterranean fever is an inherited condition characterised by recurrent episodes of painful inflammation in the abdomen, chest, or joints. These episodes are often accompanied by fever and sometimes a rash or headache. The absence of abdominal pain makes this a less likely diagnosis. Approximately 90 percent of individuals with FMF experience abdominal symptoms that can range from mild bloating to peritonitis. Although this patient is from the Mediterranean, the overall features are more in keeping with adult-onset Still's Disease.

**Hemophagocytic lymphohistiocytosis (HLH)** is incorrect. HLH is a condition in which the body makes too many activated immune cells (macrophages and lymphocytes). Clinical features include fever, hepatosplenomegaly, cytopenias, hepatitis and neurological abnormalities. Although this remains within the differential diagnosis (especially with such a high ferritin level), the absence of cytopenias and organomegaly make this a less likely diagnosis.

**Psoriatic arthritis** is incorrect. The rash described in this case is not typical of psoriasis. Psoriasis is characterised by clearly defined, red and scaly plaques.

**Rheumatoid arthritis** is incorrect. Rheumatoid nodules, accelerated nodulosis, rheumatoid vasculitis, and neutrophilic dermatosis are some of the classic lesions found in rheumatoid arthritis. The maculopapular rash in this case is not typical of rheumatoid arthritis.



Next question >

### Epidemiology

• has a bimodal age distribution - 15-25 yrs and 35-46 yrs

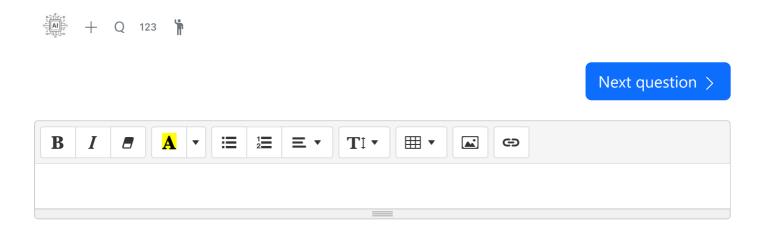
#### **Features**

- arthralgia
- elevated serum ferritin
- rash: salmon-pink, maculopapular
- pyrexia
  - typically rises in the late afternoon/early evening in a daily pattern and accompanies a worsening of joint symptoms and rash
- lymphadenopathy
- rheumatoid factor (RF) and anti-nuclear antibody (ANA) negative

The diagnosis of Still's disease in adults can be challenging. The Yamaguchi criteria is the most widely used criteria and has a sensitivity of 93.5%.

# Management

- NSAIDs
  - should be used first-line to manage fever, joint pain and serositis
  - they should be trialled for at least a week before steroids are added.
- steroids
  - o may control symptoms but won't improve prognosis
- if symptoms persist, the use of methotrexate, IL-1 or anti-TNF therapy can be considered





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49	×
50	×

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Question 33 of 178





A 52-year-old woman attends with progressive shortness of breath on exertion. On examination, you note fine bibasal inspiratory crepitations and thickening of the skin of her proximal limbs. Observations are as follows: heart rate 95 beats per minute, respiratory rate 22 breaths per minute, blood pressure 155/90 mmHg, temperature 37.5°C, and oxygen saturation 94% on air.

A high-resolution CT (HRCT) is performed:

HRCT

Ground glass changes present in the lower lobes

What antibody is associated with the development of severe interstitial lung disease?

ANA	
Anti-Jo-1	
Anti-centromere antibodies	
Anti-scl-70	
Rheumatoid factor	

Submit answer

Reference ranges ∨

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Question 33 of 178



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A high-resolution CT (HRCT) is performed:

HRCT

Ground glass changes present in the lower lobes

What antibody is associated with the development of severe interstitial lung disease?



Anti-scl-70 antibodies are associated with a higher risk of severe interstitial lung disease in systemic sclerosis

Important for me Less important



**Anti-scl-70** is correct. The clinical presentation is of diffuse cutaneous systemic sclerosis with interstitial lung disease (ILD). The most specific antibody for this subtype is anti Scl-70. This antibody is also associated with a higher risk of severe ILD.

**ANA** is incorrect. Although ANA is most often positive (>90%) it is not specific to diffuse cutaneous systemic sclerosis. Other conditions associated with a positive ANA include systemic lupus erythematosus (SLE), rheumatoid arthritis (RA), Sjogren's syndrome, Addison Disease, and autoimmune hepatitis. A positive ANA is not directly related to the development of ILD.

**Anti-Jo-1** is incorrect. This antibody is relatively specific to polymyositis and dermatomyositis. In these conditions, there are higher rates of ILD associated with a positive anti-Jo-1. However, the clinical features in this case are more suggestive of systemic sclerosis.

**Anti-centromere antibodies** is incorrect. Anti-centromere antibodies are associated with limited cutaneous systemic sclerosis which is characterised by skin thickening distal to the elbows, distal to the knees, and/or face without trunk involvement. Interstitial lung disease is not common in the limited subtype.

**Rheumatoid factor** is incorrect. Rheumatoid factor is positive in approximately 30% of cases of systemic sclerosis, however, it is not specific to this condition. The development of ILD is not linked to a positive rheumatoid factor result.



Next question >

# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - patients with renal disease should be started on an ACE inhibitor
- poor prognosis

Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







# **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis

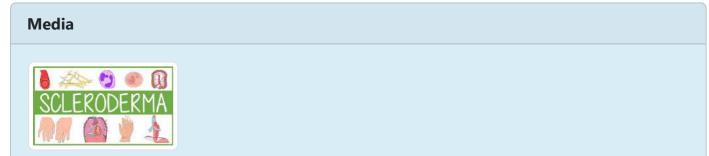


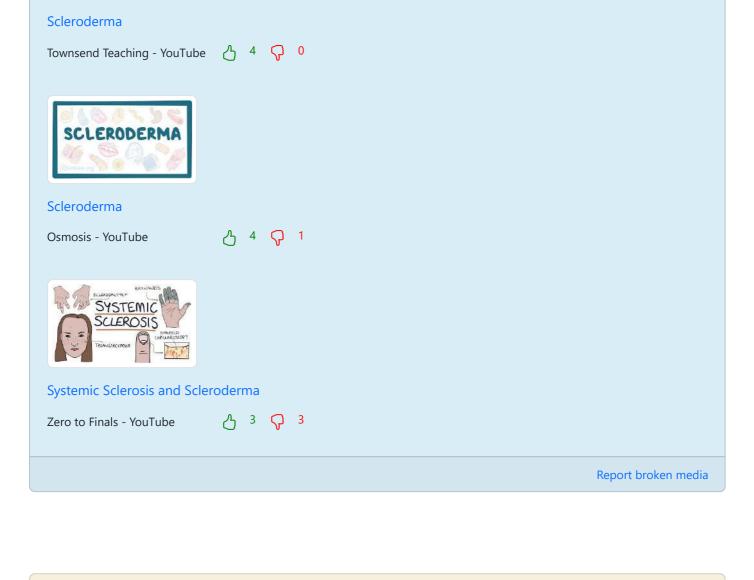
Next question >

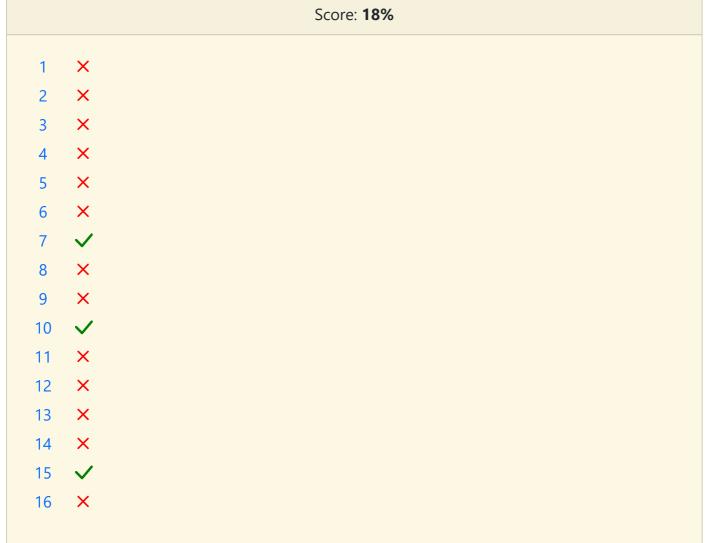












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Question 34 of 178





A 65-year-old man presents to the emergency department with visual loss in his right eye since he woke up. He states he has felt very run down over the last week with fatigue and a headache.

He is still a working farmer and has been struggling recently as two of his employees are off sick. On questioning, he complains of aching shoulders over the last 2 weeks, particularly in the morning, which he thinks may be from an increased workload.

On examination, vision in the right eye is reduced to finger counting. Vision in his left eye is 18/20.

What is the most likely fundoscopy finding on examination of his right eye?

Increased vertical cup-to-disc ratio	
Multiple abnormal vessel formation with haemorrhage	
Retinal detachment	
Swollen pale disc and blurred margins	
Venous tortuosity and flame-shaped haemorrhages	

Submit answer

Reference ranges ∨

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Score: 0%

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Question 34 of 178



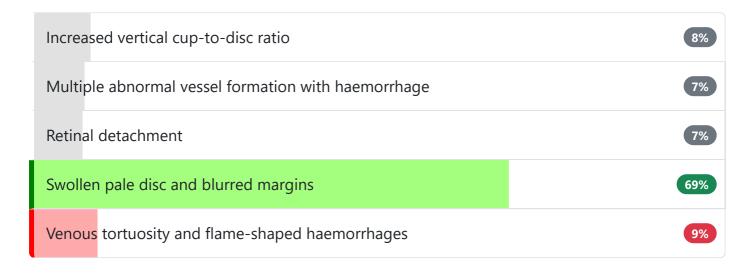


A 65-year-old man presents to the emergency department with visual loss in his right eye since he woke up. He states he has felt very run down over the last week with fatigue and a headache.

He is still a working farmer and has been struggling recently as two of his employees are off sick. On questioning, he complains of aching shoulders over the last 2 weeks, particularly in the morning, which he thinks may be from an increased workload.

On examination, vision in the right eye is reduced to finger counting. Vision in his left eye is 18/20.

What is the most likely fundoscopy finding on examination of his right eye?



Anterior ischemic optic neuropathy - fundoscopy typically shows a swollen pale disc and blurred margins

Important for me Less important



This patient has polymyalgia rheumatica (PMR), which classically presents with symmetrical pain and stiffness around the shoulders (most common), hips or neck. Symptoms are often worse in the morning. PMR is associated with temporal arteritis.

Symptoms of temporal arteritis (also known as giant cell arteritis) are constitutional upset (e.g. fatigue, fever), headache, jaw claudication and ocular involvement. This can cause permanent visual loss, of which the majority of cases are due to anterior ischaemic optic neuropathy. This occurs due to the arteritis reducing the perfusion to the optic nerve.

**Swollen pale disc and blurred margins** is the appearance of anterior ischemic optic neuropathy on fundoscopy, so this is the correct answer.

An **increased vertical cup-to-disc ratio** is classic for glaucoma. In addition, glaucoma would not explain the patient's other symptoms.

**Multiple abnormal vessel formation with haemorrhage** is typical of proliferative diabetic retinopathy where vascular endothelial growth factor (VEGF) drives the formation of new abnormal vessels.

**Retinal detachment** occurs following a retinal tear. This finding is not associated with temporal arteritis or anterior ischemic optic neuropathy.

**Venous tortuosity and flame-shaped haemorrhages** is associated with central retinal vein occlusion. Changes in temporal arteritis are largely arterial.



Next question >

# Temporal arteritis \*

Temporal arteritis (also known as giant cell arteritis: GCA) is a vasculitis of unknown cause that affects medium and large-sized vessels arteries. It occurs in those over 50 years old, with a peak incidence in patients who are in their 70s.

It requires early recognition and treatment to minimize the risk of complications such as permanent loss of vision. Hence, when temporal arteritis is suspected, treatment must be started promptly with high-dose prednisolone as well as urgent referral for assessment by a specialist.

There is an overlap between temporal arteritis and polymyalgia rheumatica (PMR) - around 50% of patients will have features of PMR.

#### **Features**

- typically patient > 60 years old
- usually rapid onset (e.g. < 1 month)</li>
- headache (found in 85%)
- jaw claudication (65%)
- vision testing is a key investigation in all patients
  - anterior ischemic optic neuropathy accounts for the majority of ocular complications. It results from occlusion of the posterior ciliary artery (a branch of the ophthalmic artery) → ischaemia of the optic nerve head. Fundoscopy typically shows a swollen pale disc and blurred margins
  - o may result in temporary visual loss amaurosis fugax
  - permanent visual loss is the most feared complication of temporal arteritis and may develop suddenly

- diplopia may also result from the involvement of any part of the oculomotor system (e.g. cranial nerves)
- tender, palpable temporal artery
- around 50% have features of PMR: aching, morning stiffness in proximal limb muscles (not weakness)
- also lethargy, depression, low-grade fever, anorexia, night sweats

#### Investigations

- raised inflammatory markers
  - ESR > 50 mm/hr (note ESR < 30 in 10% of patients)
  - o CRP may also be elevated
- temporal artery biopsy
  - skip lesions may be present
- note creatine kinase and EMG normal

#### **Treatment**

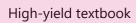
- urgent high-dose glucocorticoids should be given as soon as the diagnosis is suspected and before the temporal artery biopsy
  - o if there is no visual loss then high-dose prednisolone is used
  - if there is evolving visual loss IV methylprednisolone is usually given prior to starting high-dose prednisolone
  - o there should be a dramatic response, if not the diagnosis should be reconsidered
- urgent ophthalmology review
  - o patients with visual symptoms should be seen the same-day by an ophthalmologist
  - visual damage is often irreversible
- other treatments
  - bone protection with bisphosphonates is required as long, tapering course of steroids is required
  - o low-dose aspirin is sometimes given to patients as well, although the evidence base supporting this is weak



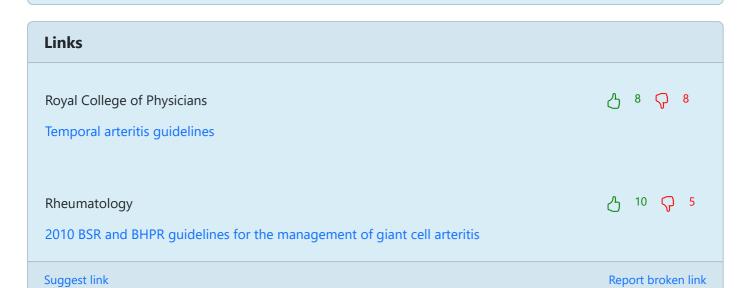
Next question >



#### **Textbooks**



#### Extended textbook



#### Media



# **Temporal Arteritis**

Pixorize - YouTube









#### Temporal arteritis

Khan Academy - YouTube









# Temporal artery biopsy

Oculoplastics.info - YouTube









Giant cell Arteritis and Takayasu arteritis (Large Vessel Vasculitis) - signs, pathophysiology

Armando Hasudungan - YouTube





Report broken media

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Question 35 of 178





An 81-year-old woman is being seen in an oncology clinic. She has been diagnosed with cancer of unknown primary with bone metastases and is currently awaiting further investigation. She has a past medical history of deep vein thrombosis and chronic kidney disease. Routine blood tests are performed and show the following:

Hb	97 g/L	(115 - 160)
Platelets	131 * 10 <sup>9</sup> /L	(150 - 400)
WBC	4.6 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	138 mmol/L	(135 - 145)
K <sup>+</sup>	4.2 mmol/L	(3.5 - 5.0)
Urea	15.9 mmol/L	(2.0 - 7.0)
Creatinine	161 µmol/L	(55 - 120)
eGFR	24 ml/min/1.73m2	(> 90)
CRP	31 mg/L	(< 5)
Vitamin D	28 ng/mL	(20-50)
Adjusted calcium	2.67 mmol/L	(2.2-2.6)

What treatment is indicated here for bone protection?

Alendronic acid	
Calcichew	×
Colecalciferol	×
Denosumab	×
No treatment required	

Submit answer

# Score: 0%





Question 35 of 178

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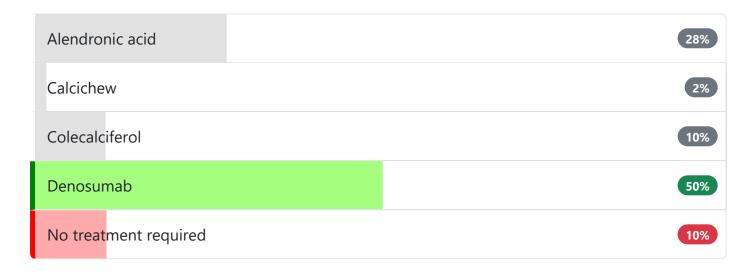
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⇒

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eGFR	24 ml/min/1.73m2	(> 90)
CRP	31 mg/L	(< 5)
Vitamin D	28 ng/mL	(20-50)
Adjusted calcium	2.67 mmol/L	(2.2-2.6)

What treatment is indicated here for bone protection?



Bisphosphonates and denosumab can be used to prevent pathological fractures in bone metastases. If the eGFR < 30, denosumab is preferred

Important for me Less important

Bone protection is indicated here due to the presence of bony metastases. This woman has an estimated glomerular filtration rate (eGFR) of approximately 25. As the eGFR is < 30, **denosumab** is preferred for bone protection over bisphosphonates such as alendronic acid.

As mentioned, bone protection is indicated here due to the presence of bony metastases however as the eGFR is < 30, denosumab would be preferred to a bisphosphonate such as **alendronic acid**.

This woman is hypercalcaemic as a result of her bony metastases. Therefore, calcium supplements such as **calcichew** are not indicated and therapies that promote osteoblastic activity are more effective for bone protection.

This woman has normal vitamin D levels and hypercalcemia. Therefore, a vitamin D analogue such as **colecalciferol** would not be indicated and therapies that promote osteoblastic activity should be prescribed instead.

As there are bony metastases here, bone protection should be prescribed. Prescribing **no treatment** would therefore be inappropriate and is incorrect.



Next question >

# Denosumab \*

Denosumab is a relatively new treatment for osteoporosis. It is a human monoclonal antibody that prevents the development of osteoclasts by <u>inhibiting RANKL</u>. Remember that osteoblasts build bone, osteoclasts eat bone. It is given as a subcutaneous injection, at a dose of 60mg, every 6 months.

A larger dose of denosumab (120mg) may also be given every 4 weeks for the prevention of skeletal-related events (i.e. pathological fractures) in adults with bone metastases from solid tumours. For example, you may have noticed some of your breast cancer patients have been prescribed denosumab.

# Where does it fit in the management of osteoporosis?

Oral bisphosphonates are still given first-line, with oral alendronate being the first-line treatment. If alendronate is not tolerated then NICE recommend using an alternative bisphosphonate - either risedronate or etidronate. Following this the advice becomes more complicated with the next-line medications only being started if certain T score and other risk factor criteria being met. Raloxifene and strontium ranelate were recommended as next-line drugs in the NICE criteria but following recent safety concerns regarding strontium ranelate it is likely there will be an increasing role for denosumab.

NICE published a technology appraisal looking at the role of denosumab in 2010. A link is

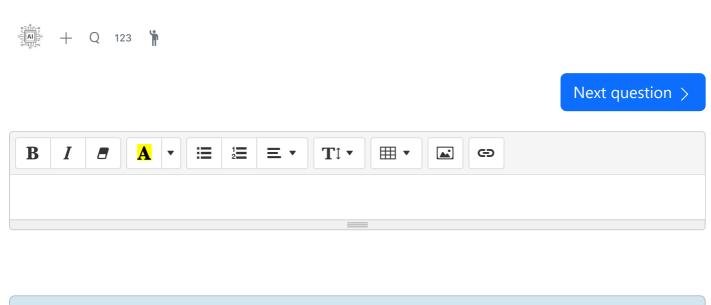
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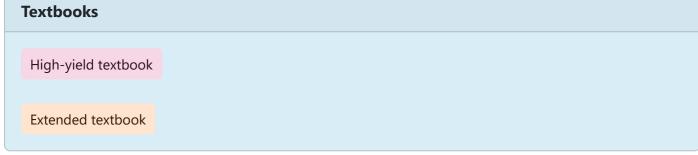
# What are the known side-effects of denosumab?

Denosumab is generally well tolerated. Dyspnoea and diarrhoea are generally considered the two most common side effects, occuring in around 1 in 10 patients. Other less common side effects include hypocalcaemia and upper respiratory tract infections.

# What does the Drug Safety Update add?

Cases of atypical femoral fractures have been noted in patients taking denosumab. Doctors are advised to look out for patients complaining of unusual thigh, hip or groin pain.







#### Media



# Osteoporosis pharmacology, prevention and treatment

Armando Hasudungan - YouTube





Report broken media

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Question 36 of 178





A 21-year-old man presents to his general practitioner with several months of low back pain. He wakes up with stiffness in the back that persists for more than an hour. There has been no obvious injury. He is a keen sportsman and finds that symptoms improve with exercise. Sitting at his office desk for prolonged periods appears to exacerbate symptoms. There is no significant past medical history except for an episode of uveitis approximately one year ago.

What would be the most appropriate first-line investigation to support the diagnosis?

Cervical X-ray	
HLA-B27	
MRI spine	
Pelvic X-ray	
Rheumatoid factor	

Submit answer

Reference ranges  $\vee$ 

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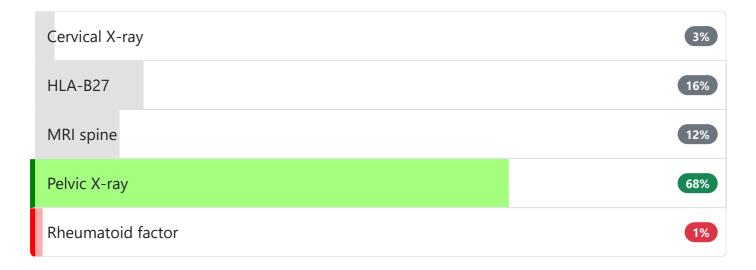


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What would be the most appropriate first-line investigation to support the diagnosis?



Diagnosis of ankylosing spondylitis can be best supported by sacro-ilitis on a pelvic X-ray

| Important for me | Less important |



This patient's history is strongly suggestive of ankylosing spondylitis, a seronegative spondyloarthropathy. It typically presents in young men; inflammatory back pain is the main clinical feature. The most important diagnostic feature is **pelvic X-ray** - the presence of sacroiliitis is a requirement for fulfilling the New York classification criteria for ankylosing spondylitis.

Whilst a **cervical X-ray** is useful to look for syndesmophytes and the 'bamboo spine', these signs are more uncommon and appear much later in the disease progression. As such, pelvic X-ray remains more useful.

**HLA-B27**, although associated with the disease in textbooks, is not particularly useful in making the diagnosis. It is not particularly specific, nor sensitive. 10% of patients with ankylosing spondylitis are HLA-B27 negative, and 10% of the healthy population are HLA-B27 positive.

A **spinal MRI** may be useful to demonstrate abnormalities when X-rays have appeared normal. However, it would not be first-line - a pelvic X-ray should be done first.

Ankylosing spondylitis is a seronegative spondyloarthropathy. Thus, testing for **rheumatoid factor** will not diagnose the condition.



Next question >

# Ankylosing spondylitis: investigation and management \*

Ankylosing spondylitis is a HLA-B27 associated spondyloarthropathy. It typically presents in males (sex ratio 3:1) aged 20-30 years old.

# Investigation

Inflammatory markers (ESR, CRP) are typically raised although normal levels do not exclude ankylosing spondylitis.

HLA-B27 is of little use in making the diagnosis as it is positive in:

- 90% of patients with ankylosing spondylitis
- 10% of normal patients

Plain x-ray of the sacroiliac joints is the most useful investigation in establishing the diagnosis. Radiographs may be normal early in disease, later changes include:

- sacroiliitis: subchondral erosions, sclerosis
- squaring of lumbar vertebrae
- 'bamboo spine' (late & uncommon)
- syndesmophytes: due to ossification of outer fibers of annulus fibrosus
- chest x-ray: apical fibrosis

If the x-ray is negative for sacroiliac joint involvement in ankylosing spondylitis but suspicion for AS remains high, the next step in the evaluation should be obtaining an MRI. Signs of early inflammation involving sacroiliac joints (bone marrow oedema) confirm the diagnosis of AS and prompt further treatment.

Spirometry may show a restrictive defect due to a combination of pulmonary fibrosis, kyphosis and ankylosis of the costovertebral joints.













# Management

The following is partly based on the 2010 EULAR guidelines (please see the link for more details):

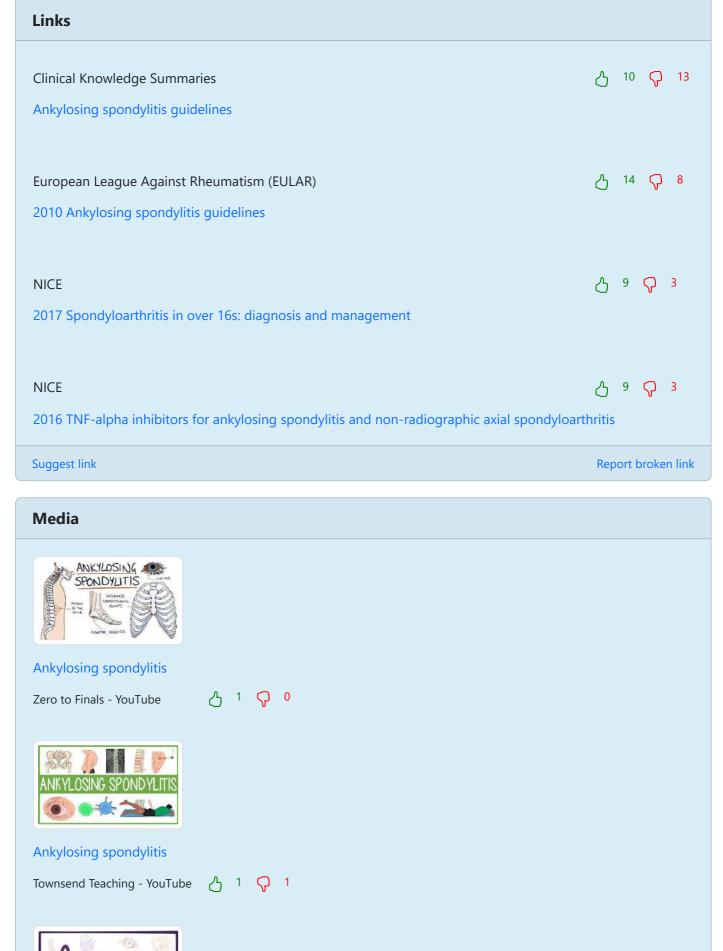
- encourage regular exercise such as swimming
- NSAIDs are the first-line treatment
- physiotherapy
- the disease-modifying drugs which are used to treat rheumatoid arthritis (such as sulphasalazine) are only really useful if there is peripheral joint involvement
- the 2010 EULAR guidelines suggest: 'Anti-TNF therapy should be given to patients with persistently high disease activity despite conventional treatments'
- research is ongoing to see whether anti-TNF therapies such as etanercept and adalimumab should be used earlier in the course of the disease



Next question >









#### Ankylosing spondylitis

Osmosis - YouTube







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Question 37 of 178





A 54-year-old woman attends with worsening shortness of breath. She has also complained of general arthralgia and severe fatigue. Her left knee had previously been red and swollen but improved with an intra-articular steroid injection. She notices around 10 minutes of early morning stiffness and then has generalised joint pains throughout the day that are worsened by over-exertion.

She has a past medical history of depression and Raynauds.

On examination, she has fine inspiratory crepitations in the lower zones. She has a reduction in her range of movement when abducting her shoulders with associated pain. The skin on her chest feels thickened. She has white nodules around her right wrist and distal interphalangeal joints.

Pulmonary function tests show:

FEV1 55% of predicted FVC 65% of predicted FEV1/FVC 0.85 DLCO 60% of predicted

Given the likely diagnosis, what is most likely to be raised in this patient?

ANCA	
Anti-Scl-70 antibodies	
Anti-centromere antibodies	
Rheumatoid factor	
Urate	

Submit answer

Reference ranges V







Question 37 of 178



 $\Box$ 



A 54-year-old woman attends with worsening shortness of breath. She has also complained of general arthralgia and severe fatigue. Her left knee had previously been red and swollen but improved with an intra-articular steroid injection. She notices around 10 minutes of early morning stiffness and then has generalised joint pains throughout the day that are worsened by over-exertion.

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On examination, she has fine inspiratory crepitations in the lower zones. She has a reduction in her range of movement when abducting her shoulders with associated pain. The skin on her chest feels thickened. She has white nodules around her right wrist and distal interphalangeal joints.

Pulmonary function tests show:

FEV1 55% of predicted FVC 65% of predicted FEV1/FVC 0.85 DLCO 60% of predicted

Given the likely diagnosis, what is most likely to be raised in this patient?

ANCA	2%
Anti-Scl-70 antibodies	76%
Anti-centromere antibodies	18%
Rheumatoid factor	4%
Urate	0%

Anti-scl-70 antibodies are associated with a higher risk of severe interstitial lung disease in systemic sclerosis

Important for me Less important



This patient has shortness of breath with fine crackles and a restrictive pattern on lung function testing. She has scleroderma over her chest and shoulders, as indicated by the thickened skin to

palpate and reduction in shoulder abduction. The white nodules on her wrist and fingers are likely calcinosis. The most likely diagnosis here is diffuse cutaneous systemic sclerosis.

**Anti-scl-70 antibodies** is correct. Anti-scl-70 antibodies are associated with diffuse cutaneous systemic sclerosis. The pattern of disease here - truncal scleroderma, respiratory involvement - is the classical findings in diffuse cutaneous systemic sclerosis rather than the limited type. The anti-scl-70 antibodies are associated with a higher risk of severe lung disease.

**Anti-centromere antibodies** is incorrect. Anti-centromere antibodies are associated with limited cutaneous systemic sclerosis. The pattern of disease here - truncal scleroderma, respiratory involvement - is the classical findings in diffuse cutaneous systemic sclerosis rather than the limited type.

**Rheumatoid factor** is incorrect. Although 30% of patients diagnosed with systemic sclerosis are rheumatoid factor positive, this patient likely has diffuse cutaneous sclerosis (given the truncal thickened skin and lung involvement). Studies have shown that up to 70% of patients with diffuse systemic sclerosis will have positive anti-scl-70 antibodies, this is, therefore, the better answer. Rheumatoid factor can also be associated with rheumatoid arthritis. Rheumatoid arthritis wouldn't explain the skin changes over her chest and shoulders, and it is less common for rheumatoid arthritis to present with monoarticular large joint effusion and exertional pain. You would expect to see an asymmetrical polyarthritis particularly in the small joints of the hands, with morning stiffness and improvement once 'getting going'.

**ANCA** is incorrect. ANCA positivity is seen in multiple autoimmune conditions, particularly vasculitis. Although this patient has some arthralgia and previous joint swelling, her other symptoms are not particularly suggestive of vasculitis. ANCA vasculitis can rarely lead to pulmonary vasculitis via progressive small-volume alveolar haemorrhage and scarring caused directly by anti-MPO antibodies, but this is unusual.

**Urate** is incorrect. Although they can appear similar, the white lesions described would be more in keeping with calcinosis than gouty tophi given the rest of the clinical picture. Gout would not explain the restrictive lung pictures or skin changes across her chest.



Next question >

# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

#### Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

#### Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - patients with renal disease should be started on an ACE inhibitor
- poor prognosis

#### Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







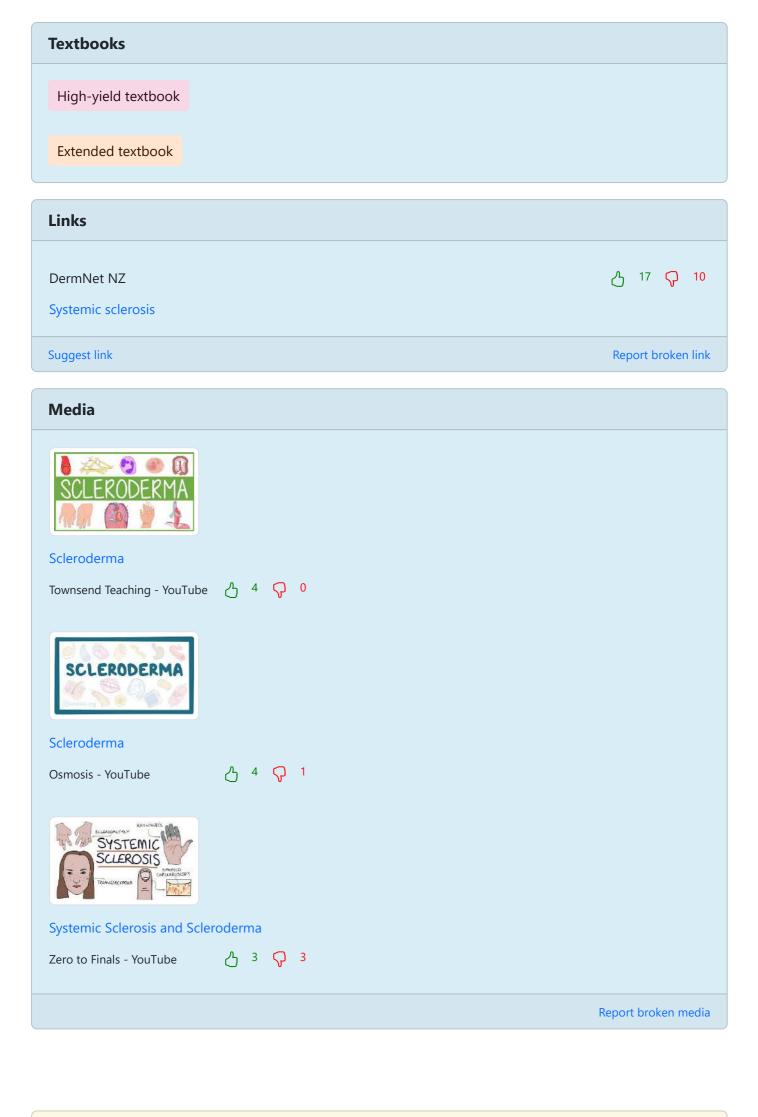
#### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis



Next question >





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Question 38 of 178





A 91-year-old woman is admitted to the hospital with an exacerbation of her chronic lower back pain and general all over body aches. A lumbosacral x-ray showed new wedge compression fractures at L4 and L5.

Bilirubin	12 µmol/L	(3 - 17)
ALP	210 u/L	(30 - 100)
ALT	20 u/L	(3 - 40)
γGT	10 u/L	(8 - 60)
Albumin	25 g/L	(35 - 50)

Calcium	1.95 mmol/L	(2.1-2.6)
Phosphate	0.8 mmol/L	(0.8-1.4)
Magnesium	0.81 mmol/L	(0.7-1.0)
Vitamin D	6 nmol/L	(>50)

What is the most appropriate management option?

40,000 units colecalciferol once weekly for 7 weeks and calcium supplements	
Give denosumab	
Start bisphosphonate and calcium supplements	
Start colecalciferol 400 units with calcium carbonate	
Start colecalciferol 800 units once daily and calcium supplements	

Submit answer

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Question 38 of 178



 $\Box$ 



A 91-year-old woman is admitted to the hospital with an exacerbation of her chronic lower back pain and general all over body aches. A lumbosacral x-ray showed new wedge compression fractures at L4 and L5.

Bilirubin	12 µmol/L	(3 - 17)
ALP	210 u/L	(30 - 100)
ALT	20 u/L	(3 - 40)
γGT	10 u/L	(8 - 60)
Albumin	25 g/L	(35 - 50)

Calcium	1.95 mmol/L	(2.1-2.6)	
Phosphate	0.8 mmol/L	(0.8-1.4)	
Magnesium	0.81 mmol/L	(0.7-1.0)	
Vitamin D	6 nmol/L	(>50)	

What is the most appropriate management option?

40,000 units colecalciferol once weekly for 7 weeks and calcium supplements	54%
Give denosumab	3%
Start bisphosphonate and calcium supplements	32%
Start colecalciferol 400 units with calcium carbonate	2%
Start colecalciferol 800 units once daily and calcium supplements	9%

Osteomalacia is managed using vitamin D supplementation (often with an initial loading dose regime)

Important for me Less important

This woman has vitamin D deficiency with a level of just 6 nmol/L. Vitamin D levels are sufficient if >50 nmol/L, insufficient if 25-50 nmol/L, and deficient if <25 nmol/L.

**40,000 units colecalciferol once weekly for 7 weeks and calcium supplements**: NICE recommends rapid replacement for vitamin D deficient patients if they are symptomatic or going

to be starting on an anti-reabsorptive agent (e.g. zoledronate, denosumab, or teriparatide). Bone and muscular pains, low mood, fatigue, and weakness are all potential symptoms of vitamin D deficiency. This patient has all over body aches and so warrants high dose loading.

**Denosumab**: denosumab is not recommended for patients with vitamin D deficiency due to the increased risk of hypocalcaemia.

**Start colecalciferol 400 units with calcium carbonate**: because this patient is symptomatic and has a vitamin D level <25 nmol/L high dose colecalciferol replacement is recommended in the first instance.

**Bisphosphonates and calcium supplements**: bisphosphonates should not be started in a vitamin D deficient patient due to the risk of precipitating hypocalcaemia. Vitamin D deficiency also decreases the effectiveness of bisphosphonates in improving bone mineral density.

**Start colecalciferol 800 units once daily and calcium supplements**: high dose colecalciferol replacement is recommended in the first instance as this patient is symptomatic of vitamin D deficiency.



Next question >

# Osteomalacia \*

Osteomalacia describes softening of the bones secondary to low vitamin D levels that in turn lead to decreased bone mineral content. If this occurs in growing children it is referred to as rickets, with the term osteomalacia preferred for adults.

#### Causes

- vitamin D deficiency
  - malabsorption
  - lack of sunlight
  - diet
- chronic kidney disease
- drug induced e.g. anticonvulsants
- inherited: hypophosphatemic rickets (previously called vitamin D-resistant rickets)
- liver disease: e.g. cirrhosis
- coeliac disease

#### **Features**

- bone pain
- bone/muscle tenderness

- fractures: especially femoral neck
- proximal myopathy: may lead to a waddling gait

#### Investigation

- bloods
  - o low vitamin D levels
  - o low calcium, phosphate (in around 30%)
  - o raised alkaline phosphatase (in 95-100% of patients)
- x-ray
  - o translucent bands (Looser's zones or pseudofractures)

#### Treatment

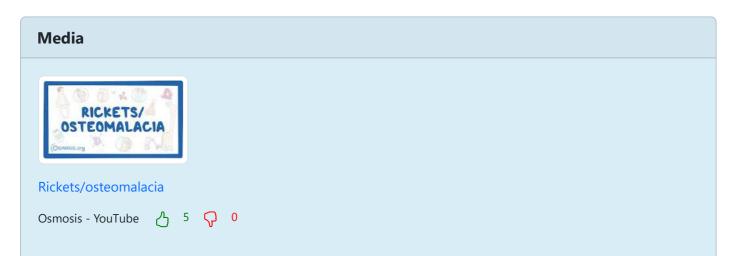
- vitamin D supplmentation
  - o a loading dose is often needed initially
- calcium supplementation if dietary calcium is inadequate



Next question >







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Question 39 of 178

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A 52-year-old lady was seen in the general medicine clinic with aches and pains. The pains were present in her arms and legs, and not associated with her joints. They have been present for several months, and she was unable to identify any precipitating factors. She also felt that on occasion she felt generally weak and tired, though denied the presence of any specific weakness. Her past medical history comprised of epilepsy which was well controlled with phenytoin 500mg BD for several years, as well as hypertension and asthma. In addition to phenytoin 500mg BD she was prescribed ramipril 5mg OD, Clenil modulite 200mcg BD, salmeterol 100mcg BD and Elleste duo for the last six months. Upon specific questioning, she stated that she ate a nutritionally balanced diet, and that she had not suffered a previous fracture. Her mother was diagnosed with osteoporosis when she was 64-years-old, and she did not smoke. She drank 10 units of alcohol per week.

On examination, she was systemically well, with a blood pressure of 132/68 mmHg, heart rate 84, respiratory rate 16/min and body mass index of 23. Examination of her cardiovascular system revealed the presence of normal heart sounds and was unremarkable. Examination of the respiratory and gastrointestinal systems was likewise unremarkable except for the presence of gingival hypertrophy. Examination of the musculoskeletal system revealed the presence of Heberden's nodes but was also otherwise unremarkable with a full range of movement in all joints. Examination of the neurological system was normal with a power of 5/5 in all muscle groups and normal sensation, tone and coordination. Cranial nerve and fundoscopy examinations were unremarkable. Examination of the thyroid gland was unremarkable.

Investigations revealed the following results:

Bilirubin	22 μmol/l
ALP	262 u/l
ALT	23 u/l
Albumin	42 g/l
Protein	76 g/l
Globulin	34 g/l
Adjusted calcium	2.06 mmol/l
Phosphate	0.78 mmol/l
Vitamin D level	pending result
Parathyroid hormone	88 (NR 11-54 pg/ml)
IgG	11.2 g/L (NR 7.0 18.0)
IgA	3.2 g/L(NR 0.8 4.0)
IgM	2.1 g/L (NR 0.4 2.5)

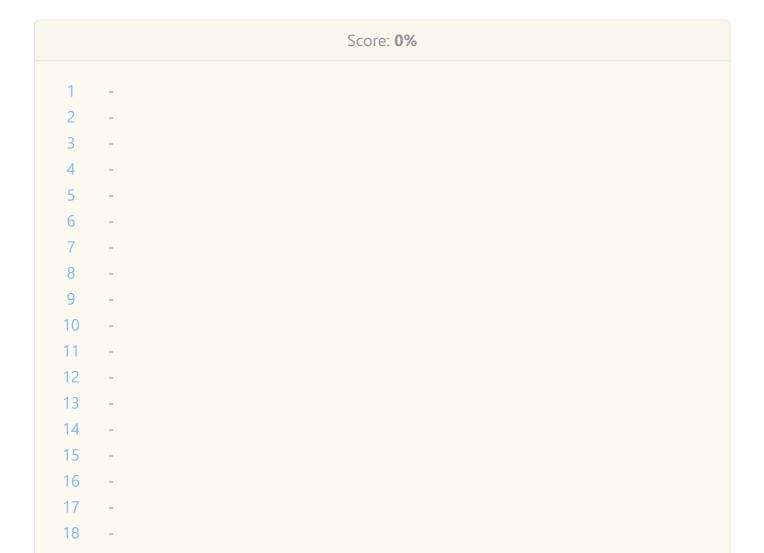
Urinary Bence Jones Protein: negative

What is the most likely underlying diagnosis?

Fibromyalgia	
Paget's disease	
Primary hyperparathyroidism	
Osteoporosis	
Osteomalacia	

# Submit answer

Reference ranges  $\vee$ 



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Question 39 of 178







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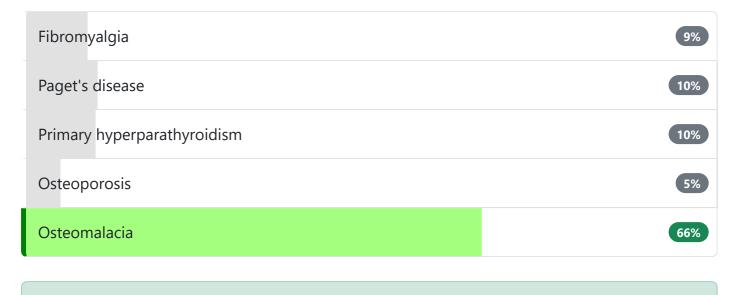
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IgG	11.2 g/L (NR 7.0 18.0)
IgA	3.2 g/L(NR 0.8 4.0)
IgM	2.1 g/L (NR 0.4 2.5)

Urinary Bence Jones Protein: negative

What is the most likely underlying diagnosis?

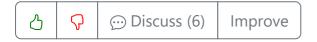


Bone pain, tenderness and proximal myopathy (→ waddling gait) → ?osteomalacia

| Important for me | Less important |



Phenytoin is known to affect the metabolism of vitamin D, and over prolonged periods of time may result in osteomalacia. This patient is also likely suffering other sequelae of prolonged phenytoin use as manifested by the presence of gingival hypertrophy. She has a raised parathyroid hormone level secondary to the low vitamin D level, and there is no evidence of osteoporosis.



Next question >

# Osteomalacia \*

Osteomalacia describes softening of the bones secondary to low vitamin D levels that in turn lead to decreased bone mineral content. If this occurs in growing children it is referred to as rickets, with the term osteomalacia preferred for adults.

#### Causes

- vitamin D deficiency
  - malabsorption
  - lack of sunlight
  - diet
- chronic kidney disease
- drug induced e.g. anticonvulsants

- inherited: hypophosphatemic rickets (previously called vitamin D-resistant rickets)
- liver disease: e.g. cirrhosis
- coeliac disease

#### **Features**

- bone pain
- bone/muscle tenderness
- fractures: especially femoral neck
- proximal myopathy: may lead to a waddling gait

#### Investigation

- bloods
  - low vitamin D levels
  - low calcium, phosphate (in around 30%)
  - o raised alkaline phosphatase (in 95-100% of patients)
- x-ray
  - o translucent bands (Looser's zones or pseudofractures)

#### **Treatment**

- vitamin D supplmentation
  - a loading dose is often needed initially
- calcium supplementation if dietary calcium is inadequate



Next question >



# Textbooks High-yield textbook Extended textbook

#### Media



#### Rickets/osteomalacia

Osmosis - YouTube  $\int_0^{\infty} 5 \quad \bigcirc 0$ 







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#### Question 40 of 178





A 34-year-old woman is reviewed in clinic. She has been suffering from increasing tiredness for the last three months. She has no other symptoms and specifically denies pain, bleeding, weight loss and mood problems. She has a past medical history of asthma but has not needed her blue inhaler in over a year. She has no allergies. Her only regular medication is the oral contraceptive pill. Blood tests show a normal FBC, U&Es, calcium, parathyroid hormone but low vitamin D (32 nmol/L). What is the most appropriate treatment in regards to her low vitamin D?

Dietary advise only	
Loading dose vitamin D	
Maintenance dose vitamin D	
Combined calcium and vitamin D	
No treatment needed	

#### Submit answer

Reference ranges ∨

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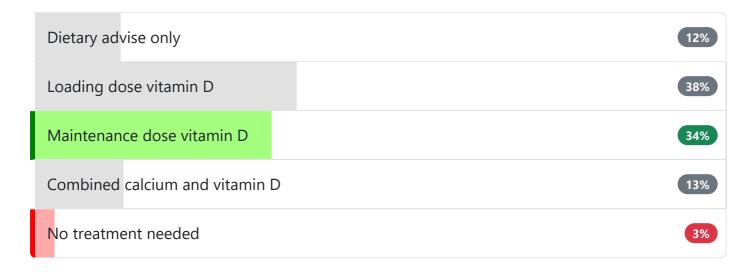
Question 40 of 178



 $\Box$ 



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Osteomalacia is managed using vitamin D supplementation (often with an initial loading dose regime)

Important for me Less important

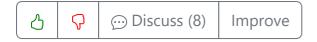
This patient has vitamin D insufficiency which may explain her tiredness. This should be managed with maintenance dose vitamin D. Loading dose would be appropriate if her serum level was less than 30 nmol/L. Dietary advice would be appropriate for patients with adequate levels, above 50 nmol/L. As calcium is normal and there is no evidence of low calcium diet there is no need for calcium supplementation.

Vitamin D deficiency versus insufficiency

Description	Serum levels	Treatment
Adequate vitamin D	>50 nmol/L	dietary recommendations
Insufficient vitamin D	30-50 nmol/L	maintenance dose vitamin D
Deficient vitamin D	<30 nmol/L	loading dose vitamin D

Source:

'Vitamin D Deficiency in Adults - Treatment and Prevention.' Clinical Knowledge Summaries. National Institute for Health and Care Excellence, Nov. 2016.



Next question >

# Osteomalacia \*

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  - o malabsorption
  - lack of sunlight
  - o diet
- chronic kidney disease
- drug induced e.g. anticonvulsants
- inherited: hypophosphatemic rickets (previously called vitamin D-resistant rickets)
- liver disease: e.g. cirrhosis
- coeliac disease

#### **Features**

- bone pain
- bone/muscle tenderness
- fractures: especially femoral neck
- proximal myopathy: may lead to a waddling gait

#### Investigation

- bloods
  - low vitamin D levels
  - low calcium, phosphate (in around 30%)
  - raised alkaline phosphatase (in 95-100% of patients)
- x-ray
  - translucent bands (Looser's zones or pseudofractures)

#### **Treatment**

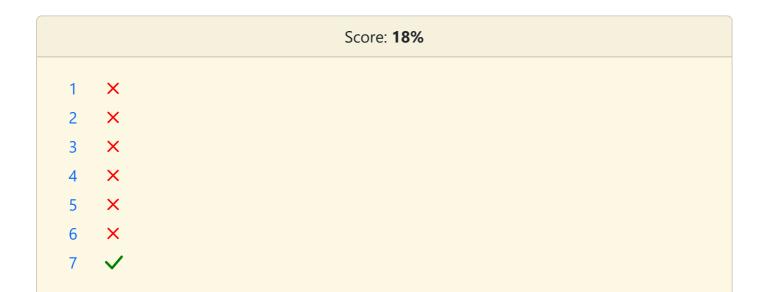
- vitamin D supplmentation
  - o a loading dose is often needed initially
- calcium supplementation if dietary calcium is inadequate

Next question >



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### Question 41 of 178

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A 75-year-old male presents to the emergency department with two episodes of loss of consciousness over the last 48 hours. Both episodes were witnessed by his wife, onset while sitting in his chair at home, without any witnessed limb jerking, urinary incontinence or tongue biting. He denies any chest pain or shortness of breath normally but reports gradually being able to walk increasingly shorter distances, which he attributed to old age. He has no other significant past medical history, lives with his wife and is a lifelong non-smoker. On examination, he has a significant thoracic kyphosis. On flexion of the lower back, the marked distance increased from 15 cm to 18 cm. He also has a poverty of spinal lateral flexion and bilateral spinal rotation. His cardiovascular examination reveals heart sounds I and II with an early diastolic murmur. Respiratory examination reveals fine inspiratory crackles at both apices. His lying and standing blood pressures are unremarkable. A CT head demonstrated only mild microangiopathic disease. The patient is currently comfortable and alert, requesting to go home. He is attached to cardiac telemetry. What do you expect his ECG to show?

Sinus bradycardia	
Trigeminy	
Fast atrial fibrillation with ventricular response greater than 100	
Atrial flutter	
Bradycardia with 1st degree heart block	

Submit answer

Reference ranges  $\vee$ 

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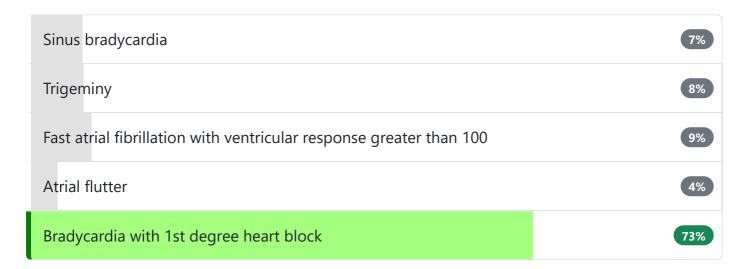


Question 41 of 178



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Ankylosing spondylitis features - the 'A's

- Apical fibrosis
- Anterior uveitis
- Aortic regurgitation
- Achilles tendonitis
- AV node block
- Amyloidosis

Important for me Less important



The clinical description is of a patient with ankylosing spondylitis, associated with multiple extraarticular features, commonly remembered by MRCP candidates as the As. They classically include atlantoaxial subluxation, arachnoiditis, apical fibrosis, anterior uveitis, aortic regurgitation, AV block, amyloidosis, IgA nephropathy, Achilles tendonitis and associations with plantar fasciitis

and inflammatory bowel disease (the last two are a bit of a stretch!). In this case, the patient is Schober's positive with a murmur of aortic regurgitation from aortitis. The most likely cause of these episodes of syncope are cardiac in origin. AV node block results in first-degree heart block on ECG, resulting in symptoms if bradycardia leads to transient cerebral hypoperfusion.



Next question >

### Ankylosing spondylitis: features \*

Ankylosing spondylitis is a HLA-B27 associated spondyloarthropathy. It typically presents in males (sex ratio 3:1) aged 20-30 years old.

### **Features**

- typically a young man who presents with lower back pain and stiffness of insidious onset
- stiffness is usually worse in the morning and improves with exercise
- the patient may experience pain at night which improves on getting up

### Clinical examination

- reduced lateral flexion
- reduced forward flexion Schober's test a line is drawn 10 cm above and 5 cm below the back dimples (dimples of Venus). The distance between the two lines should increase by more than 5 cm when the patient bends as far forward as possible
- reduced chest expansion

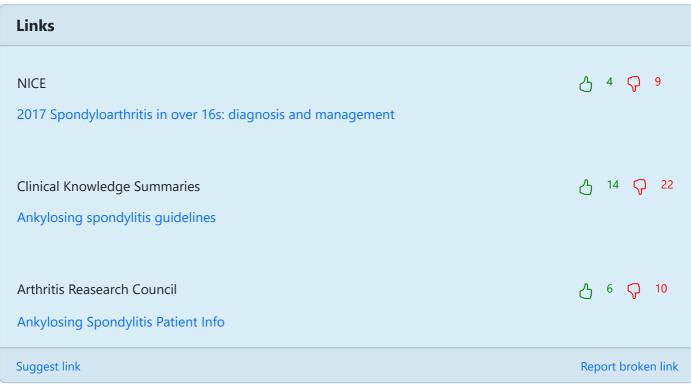
### Other features - the 'A's

- Apical fibrosis
- Anterior uveitis
- Aortic regurgitation
- Achilles tendonitis
- AV node block
- Amyloidosis
- and cauda equina syndrome
- peripheral arthritis (25%, more common if female)













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Question 42 of 178





A 71-year-old woman is reviewed in the oncology clinic. She has a past medical history of metastatic breast cancer, hypertension and chronic kidney disease. She is treated with doxorubicin, cyclophosphamide, amlodipine and ramipril. She does not smoke or drink alcohol. She lives with her husband and is a retired nurse.

Examination reveals evidence of bilateral mastectomies but is otherwise unremarkable.

### Blood tests:

Hb	121 g/L	Male: (135-180) Female: (115 - 160)
Platelets	489 * 10 <sup>9</sup> /L	(150 - 400)
WBC	8.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	136 mmol/L	(135 - 145)
K <sup>+</sup>	4.2 mmol/L	(3.5 - 5.0)
Urea	21.4 mmol/L	(2.0 - 7.0)
Creatinine	234 μmol/L	(55 - 120)
eGFR	27 mL/min/1.73m2	(>90)
CRP	44 mg/L	(< 5)
Bilirubin	14 μmol/L	(3 - 17)
ALP	189 u/L	(30 - 100)
ALT	28 u/L	(3 - 40)
γGT	42 u/L	(8 - 60)
Albumin	31 g/L	(35 - 50)
Calcium	2.32 mmol/L	(2.20-2.6)

A recent staging CT scan demonstrates bony metastatic disease in the vertebral column.

Based on the clinical history, what is the most appropriate medication to prevent pathological fractures?

Alendronic acid	
Denosumab	
Hormone replacement therapy (HRT)	

Teriparatide	
Zoledronic acid	

### Submit answer

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Question 42 of 178







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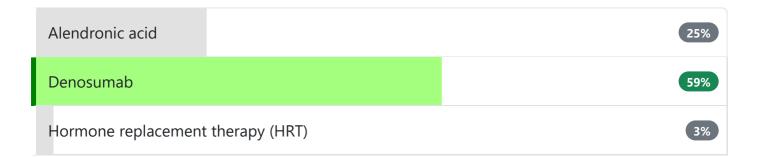
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Albumin	31 g/L	(35 - 50)
Calcium	2.32 mmol/L	(2.20-2.6)

A recent staging CT scan demonstrates bony metastatic disease in the vertebral column.

Based on the clinical history, what is the most appropriate medication to prevent pathological fractures?





Bisphosphonates and denosumab can be used to prevent pathological fractures in bone metastases. If the eGFR < 30, denosumab is preferred

Important for me Less important

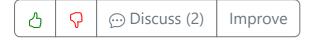
Denosumab is the correct answer. This can be used to prevent pathological fractures in those patients with bone metastases. It is the preferred option when the estimated glomerular filtration rate is between 20-30, as in this case.

Alendronic acid is incorrect. This is an option for preventing pathological fractures in bone metastases but is contraindicated when the estimated glomerular filtration rate is < 30.

Zoledronic acid is incorrect. This is an option for preventing pathological fractures in bone metastases but is contraindicated when the estimated glomerular filtration rate is < 30.

Teriparatide is incorrect. This medication is contraindicated in the presence of bone metastases.

HRT is incorrect. This is not a medication used to prevent pathological fracture and is associated with increased breast cancer risk.



Next question >

### Denosumab \*

Denosumab is a relatively new treatment for osteoporosis. It is a human monoclonal antibody that prevents the development of osteoclasts by <u>inhibiting RANKL</u>. Remember that osteoblasts build bone, osteoclasts eat bone. It is given as a subcutaneous injection, at a dose of 60mg, every 6 months.

A larger dose of denosumab (120mg) may also be given every 4 weeks for the prevention of skeletal-related events (i.e. pathological fractures) in adults with bone metastases from solid tumours. For example, you may have noticed some of your breast cancer patients have been prescribed denosumab.

Where does it fit in the management of osteoporosis?

Oral bisphosphonates are still given first-line, with oral alendronate being the first-line treatment. If alendronate is not tolerated then NICE recommend using an alternative bisphosphonate - either risedronate or etidronate. Following this the advice becomes more complicated with the next-line medications only being started if certain T score and other risk factor criteria being met. Raloxifene and strontium ranelate were recommended as next-line drugs in the NICE criteria but following recent safety concerns regarding strontium ranelate it is likely there will be an increasing role for denosumab.

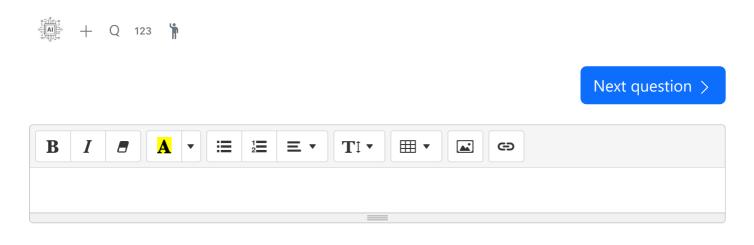
NICE published a technology appraisal looking at the role of denosumab in 2010. A link is provided.

### What are the known side-effects of denosumab?

Denosumab is generally well tolerated. Dyspnoea and diarrhoea are generally considered the two most common side effects, occuring in around 1 in 10 patients. Other less common side effects include hypocalcaemia and upper respiratory tract infections.

### What does the Drug Safety Update add?

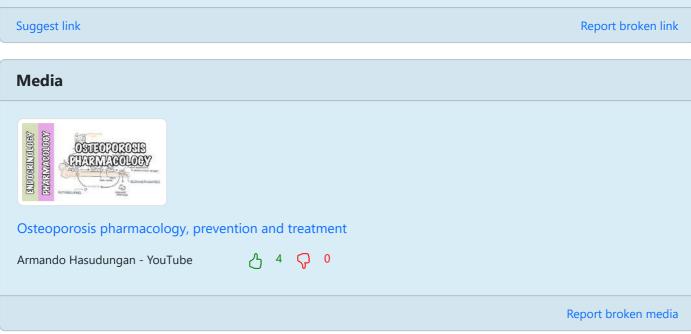
Cases of atypical femoral fractures have been noted in patients taking denosumab. Doctors are advised to look out for patients complaining of unusual thigh, hip or groin pain.

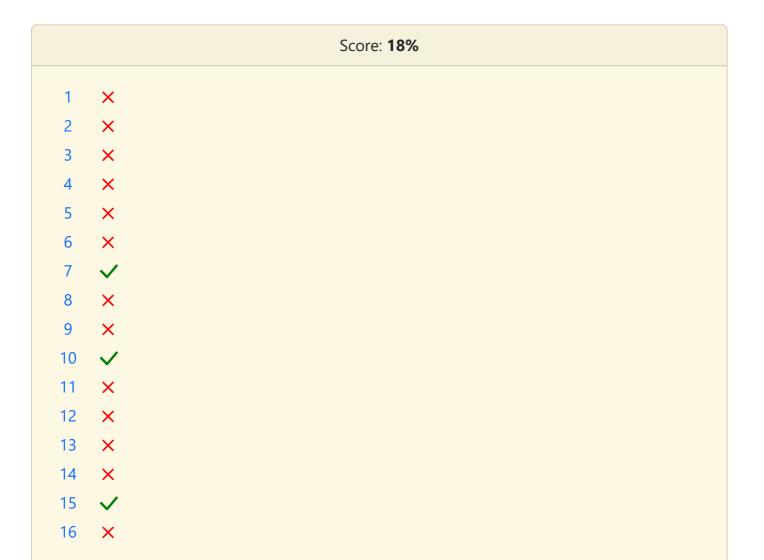




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Question 43 of 178





A 72-year-old woman attends the clinic with widespread joint pain. On examination, she has prominent synovitis of her proximal interphalangeal (PIP) and metacarpophalangeal (MCP) joints of the hands, the wrists, and small joints of the feet including the metatarsophalangeal (MTP) joints.

### Blood results are as follows:

Hb	110 g/L	Male: (135-180) Female: (115 - 160)
Platelets	524 * 10 <sup>9</sup> /L	(150 - 400)
WBC	12.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	135 mmol/L	(135 - 145)
K <sup>+</sup>	3.6 mmol/L	(3.5 - 5.0)
Urea	6.8 mmol/L	(2.0 - 7.0)
Creatinine	92 μmol/L	(55 - 120)
CRP	2 mg/L	(< 5)

Bilirubin	14 µmol/L	(3 - 17)
ALP	34 u/L	(30 - 100)
ALT	22 u/L	(3 - 40)
γGT	42 u/L	(8 - 60)
Albumin	34 g/L	(35 - 50)

A decision is made to start her on methotrexate.

What monitoring blood tests are required?

Full blood count and renal and liver function in 1 week	
Full blood count and renal and liver function in 4 weeks	
Full blood count in 1 week	
Full blood count, bone profile and renal and liver function in 1 week	
Full blood count, bone profile and renal and liver function in 4 weeks	

### Submit answer

Reference ranges  $\vee$ 

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Question 43 of 178







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ALT	22 u/L	(3 - 40)	
γGT	42 u/L	(8 - 60)	
Albumin	34 g/L	(35 - 50)	

A decision is made to start her on methotrexate.

What monitoring blood tests are required?

Full blood count and renal and liver function in 1 week	49%
Full blood count and renal and liver function in 4 weeks	38%
Full blood count in 1 week	3%
Full blood count, bone profile and renal and liver function in 1 week	5%
Full blood count, bone profile and renal and liver function in 4 weeks	5%

**Full blood count and renal and liver function in 1 week** is correct. Patients who have started methotrexate should have full blood count and renal and liver function tests repeated every 1-2 weeks until therapy stabilised. Thereafter patients should be monitored every 2-3 months.

**Full blood count and renal and liver function in 4 weeks** is incorrect. The BNF recommends that they are checked every 1-2 weeks until therapy is stabilised.

**Full blood count in 1 week** is incorrect. The BNF recommends that liver and renal function are also monitored.

**Full blood count, bone profile and renal and liver function in 1 week** is incorrect. There is no requirement to monitor bone profile.

**Full blood count, bone profile and renal and liver function in 4 weeks** is incorrect. There is no requirement to monitor bone profile. Furthermore, the BNF recommends that they are checked every 1-2 weeks until therapy is stabilised.



Next question >

### Methotrexate ★

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

### **Indications**

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - the most common pulmonary manifestation

- similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
- o typically develops within a year of starting treatment, either acutely or subacutely
- o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

### Prescribing methotrexate

- methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

### Interactions

- avoid prescribing <u>trimethoprim</u> or <u>co-trimoxazole</u> concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

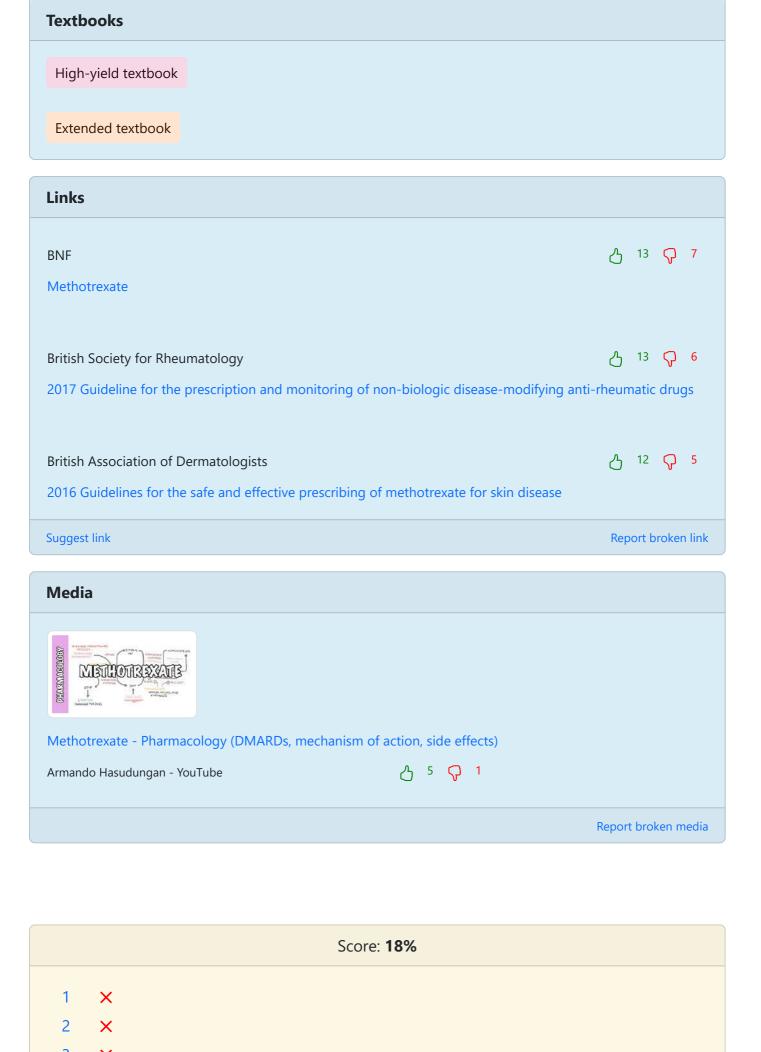
### Methotrexate toxicity

the treatment of choice is folinic acid



Next question >





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Question 44 of 178

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A 72-year-old male from the Dominican Republic presents with a 4-month history of constant bilateral anterior thigh pain and lower limb weakness, starting first on the left before progressing to bilateral symptoms. He reports being an extremely active man prior to these symptoms, continually to work as a landscape gardener until four months ago, and now he is wheelchair and bed-bound. He denies any history of trauma to his hips, previous tuberculosis infection or contact with heavy metals. His thigh pain appears excruciating, described as burning in nature along the anterior aspects of both thighs to the origin of the patella tendon bilaterally. He reports no recent weight loss, pyrexia or skin changes. His past medical history includes hypertension, hypercholesterolaemia, hypothyroidism and type 2 diabetes mellitus (diagnosed 6 years ago).

On examination, the patient has mildly reduced muscle bulk in bilateral hip flexors and bilateral fasciculations in both thighs. Examination of power demonstrates 2+/5 bilaterally in hip flexion 4+/5 hip extension bilaterally and 5/5 all other lower limb movements. Sensory examination revealed reduced sensation to cotton wool in both thighs and distal feet, reduced proprioception in both toes. Reflexes were 2+ at both knee jerks, absent ankle jerks bilaterally and downgoing plantars bilaterally. His blood tests are as follows:

Hb	134 g/l
Platelets	383 * 10 <sup>9</sup> /l
WBC	4.5 * 10 <sup>9</sup> /l

Na <sup>+</sup>	135 mmol/l
K <sup>+</sup>	4.6 mmol/l
Urea	8.6 mmol/l
Creatinine	112 µmol/l
CRP	1 mg/l
Creatine kinase	116 IU/I (50-335)
HbA1c	68 mmol/mol
TSH	2.1 mu/l
Free T4	15.4 nmol/l

An MRI scan was performed of his lumbosacral plexus, demonstrating no appreciable structural lesion. Nerve conduction studies and EMG are awaited.

What is the most optimal treatment?

Optimise diabetic control	
Riluzole	×
Intravenous immunoglobulin	×
Intravenous methylprednisolone	×
Lumbar puncture	

### Submit answer

Reference ranges  $\checkmark$ 

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Question 44 of 178



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TSH	2.1 mu/l
Free T4	15.4 nmol/l

An MRI scan was performed of his lumbosacral plexus, demonstrating no appreciable structural lesion. Nerve conduction studies and EMG are awaited.

What is the most optimal treatment?

Optimise diabetic control	38%
Riluzole	17%
Intravenous immunoglobulin	16%
Intravenous methylprednisolone	27%
Lumbar puncture	2%

The positive symptoms include bilateral proximal myopathy, severe neuropathic pain in thighs and absence of lumbosacral structural lesions, in a HIV negative patient, the diagnosis if most likely diabetic amyotrophy. The onset of proximal pain as a result of a microvascular lumbosacral plexopathy is usually asymmetric before becoming more symmetrical.

The lack of upper motor neurone signs makes this unlikely to be motor neuron disease (and hence requiring riluzole). The involvement of sensory components rules out multifocal motor neuropathy with conduction block, typically responsive to IVIg. The proximal location of the symptoms and prominent neuropathic pain is classical of amyotrophy instead of chronic inflammatory demyelinating polyneuropathy (CIDP), for which intravenous methylprednisolone can be therapeutic.

The best treatment for diabetic amyotrophy is to optimise blood sugar control and symptomatic treatment neuropathic agents. Most patients report at least partial, if not full motor recovery and much-improved pain symptoms.



Next question >

### Diabetic amyotrophy \*

Diabetic amyotrophy is also known as proximal diabetic neuropathy. The latter term is probably more useful as it describes more accurately the aetiology of the condition.

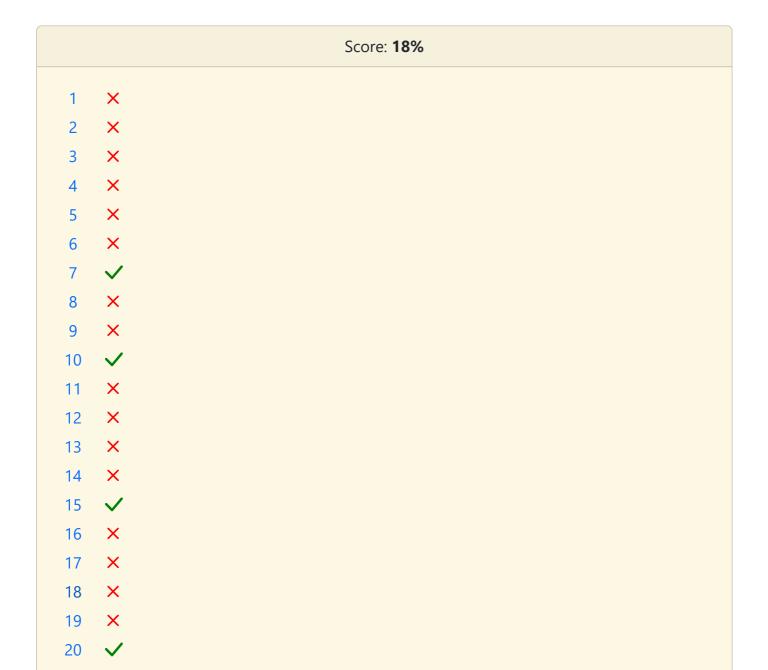
Typical features include

- pain is usually in the first symptom, often in the hips or buttocks
- this is followed by weakness, for example difficulty getting out of a chair





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### Question 45 of 178

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 $\overline{\Rightarrow}$ 

A 23-year-old comes in with a painful right knee. He denies any specific trauma. He has no past medical history. On examination, he has a red and warm knee with a moderate effusion. His observations show a respiratory rate of 24/min, blood pressure 120/72 mmHg, temperature 37.8°C. His knee is aspirated which is cloudy in appearance. Laboratory testing shows calcium pyrophosphate crystals and the Gram stain is awaited. What is the most appropriate treatment plan?

Admit for intravenous antibiotics	
Home with analgesia	
Admit awaiting cultures	
Check ferritin	
Intra-articular depo-medrone	

Submit answer

Reference ranges  $\vee$ 

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Question 45 of 178

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Septic arthritis has not been ruled out until there are no organisms seen. The patient should be treated with intravenous antibiotics as soon as the aspirate has been taken and only be discharged once it is confirmed that they do not have septic arthritis.

Although calcium pyrophosphate crystals are most associated with pseudo gout they do not rule out septic arthritis. The ferritin would be raised in septic arthritis so using it as a screen for haemochromatosis would not be advisable in this setting.



Next question >

### Septic arthritis in adults \*

### Pathophysiology

- most common organism overall is Staphylococcus aureus
  - o in young adults who are sexually active, *Neisseria gonorrhoeae* is the most common organism (disseminated gonococcal infection)
- the most common cause is hematogenous spread
  - this may be from distant bacterial infections e.g. abscesses
- in adults, the most common location is the knee

### **Features**

- acute, swollen joint
  - o restricted movement in 80% of patients
  - examination findings: warm to touch/fluctuant
- fever: present in the majority of patients

### Investigations

- synovial fluid sampling is obligatory
  - this should be done prior to the administration of antibiotics if necessary
  - o may need to be done under radiographic guidance
  - shows a leucocytosis with neutrophil predominance
  - o gram staining is negative in around 30-50% of cases
  - o fluid culture is positive in patients with non-gonococcal septic arthritis
- blood cultures: the most common cause of septic arthritis is hematogenous spread
- joint imaging

### Management

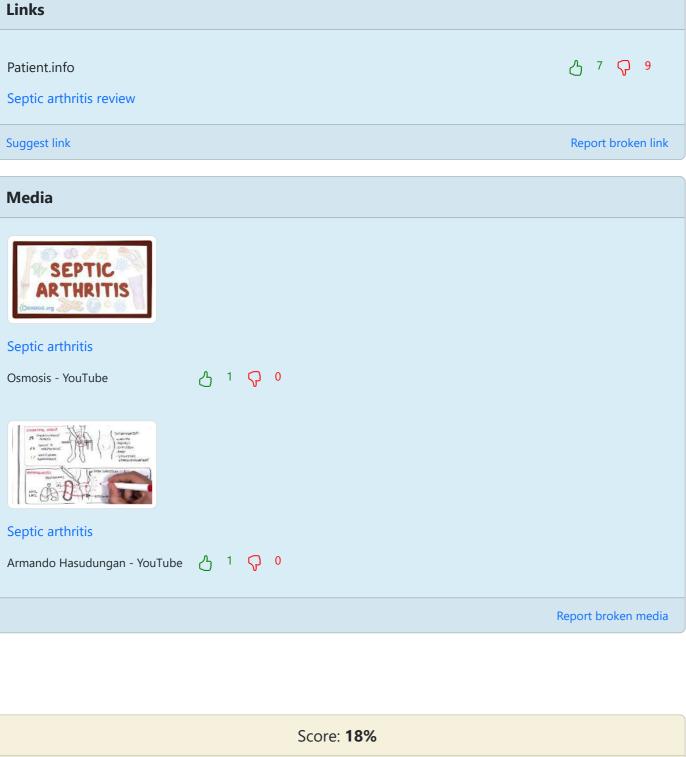
- intravenous antibiotics which cover Gram-positive cocci are indicated. The BNF currently recommends flucloxacillin or clindamycin if penicillin allergic
  - o antibiotic treatment is normally be given for several weeks (BNF states 4-6 weeks)
  - o patients are typically switched to oral antibiotics after 2 weeks
- needle aspiration should be used to decompress the joint
- arthroscopic lavage may be required

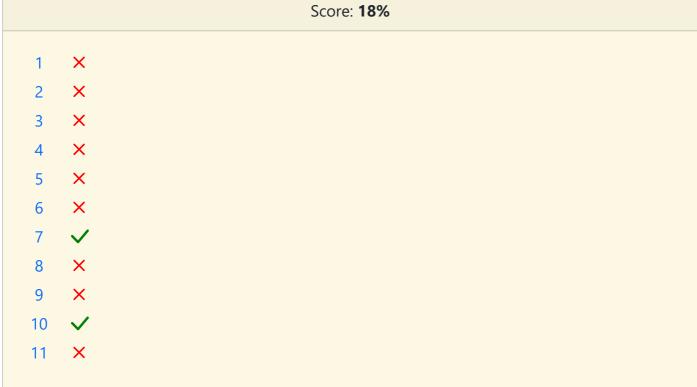


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A 55-year-old woman was called by an out of hours GP and told to attend the emergency department due to abnormalities found on her blood tests which were taken at her annual well woman's check-up.

Her past medical history includes systemic sclerosis. She uses topical emollients on her hands and has no allergies. She works as a receptionist, is an ex-smoker and denies alcohol consumption. Her blood results are shown below:

Hb	110 g/L	(115 - 160)
Platelets	151 * 10 <sup>9</sup> /L	(150 - 400)
WBC	4.5 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	137 mmol/L	(135 - 145)
K <sup>+</sup>	4.2 mmol/L	(3.5 - 5.0)
Urea	11.5 mmol/L	(2.0 - 7.0)
Creatinine	164 µmol/L	(55 - 120)
eGFR	30 ml/min	(>90)

The nurse does her observations and informs you her blood pressure is high. In the right arm, it is 187/95mmHg and in the left arm is 191/94mmHg.

What would be the most appropriate treatment to initiate in this patient?

Lisinopril	
Amlodipine	
Bendroflumethiazide	
Doxazosin	
Labetalol	

Submit answer

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 $\Box$ 



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Urea	11.5 mmol/L	(2.0 - 7.0)
Creatinine	164 µmol/L	(55 - 120)
eGFR	30 ml/min	(>90)

The nurse does her observations and informs you her blood pressure is high. In the right arm, it is 187/95mmHg and in the left arm is 191/94mmHg.

What would be the most appropriate treatment to initiate in this patient?

Lisinopril	81%
Amlodipine	11%
Bendroflumethiazide	1%
Doxazosin	2%
Labetalol	5%

Renal complications of systemic sclerosis - ACE-inhibitors

Important for me Less important



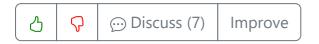
Renal complications of system sclerosis often present with hypertension and AKI. ACE inhibitors are used 1st line in this instance, therefore lisinopril is the correct choice. A severe sclerodermic renal crisis may also present with microangiopathic haemolytic anaemia.

Amlodipine is a calcium channel blocker that is used in the management of hypertension.

Bendroflumethiazide is a thiazide diuretic that can also be used to manage hypertension.

Doxazosin is an  $\alpha$  blocker used to treat hypertension.

Labetalol is a  $\beta$  blocker. It is commonly used to acutely lower blood pressure in haemorrhagic strokes.



Next question >

# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - patients with renal disease should be started on an ACE inhibitor
- poor prognosis

Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







#### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis



Next question >







#### Media



#### Scleroderma

Townsend Teaching - YouTube 4 Q 0









#### Scleroderma

Osmosis - YouTube









#### Systemic Sclerosis and Scleroderma

Zero to Finals - YouTube







Report broken media

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#### Question 47 of 178





A 45-year-old female attends the acute medical unit with fever and rigors. Her past medical history includes rheumatoid arthritis. Her regular medicines include methotrexate and sulfasalazine. She was recently treated with trimethoprim for a urinary tract infection.

#### Blood results are as follows:

Hb	110 g/l	Na <sup>+</sup>	138 mmol/l
Platelets	94 * 10 <sup>9</sup> /l	K <sup>+</sup>	3.8 mmol/l
WBC	1.2 * 10 <sup>9</sup> /l	Urea	7.8 mmol/l
Neuts	0.6 * 10 <sup>9</sup> /l	Creatinine	104 µmol/l
Lymphs	0.4 * 10 <sup>9</sup> /l	CRP	212 mg/l

#### What treatment is indicated?

Platelet transfusion	
Folinic acid	
Folate	
Sodium bicarbonate	
Reassurance	

# Submit answer

Reference ranges ∨

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Question 47 of 178



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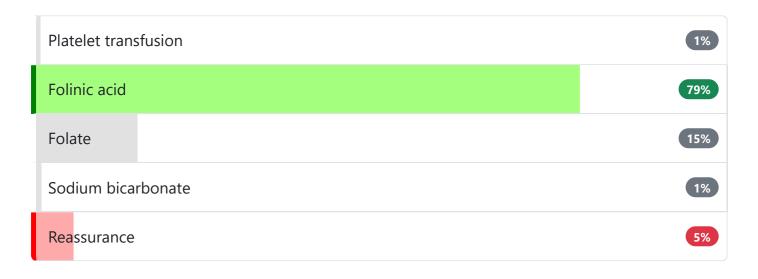


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Neuts	0.6 * 10 <sup>9</sup> /l	Creatinine	104 µmol/l
Lymphs	0.4 * 10 <sup>9</sup> /l	CRP	212 mg/l

#### What treatment is indicated?



Folinic acid is the treatment of choice for methotrexate toxicity

Important for me Less important



The patient is on methotrexate which is a dihydrofolate reductase antagonist. Trimethoprim is a selective inhibitor of dihydrofolate reductase and is therefore absolutely contraindicated in patients who take methotrexate. The patient has developed bone marrow suppression secondary to methotrexate toxicity, and neutropenic sepsis.

Folinic acid can bypass the effect of methotrexate on dihydrofolate reductase and replete the intracellular supply of folate. The patient should, therefore, receive folinic acid urgently.

A platelet transfusion is generally reserved for cases when the platelet count falls below  $50 * 10^9$ /l.

In addition, the patient should be treated for neutropenic sepsis with broad-spectrum antibiotics.

Urinary alkalisation with sodium bicarbonate has been shown to enhance drug excretion, however the evidence is limited.



Next question >

# Methotrexate **★**

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

#### Indications

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

#### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - o typically develops within a year of starting treatment, either acutely or subacutely
  - o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

#### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

#### Prescribing methotrexate

• methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use

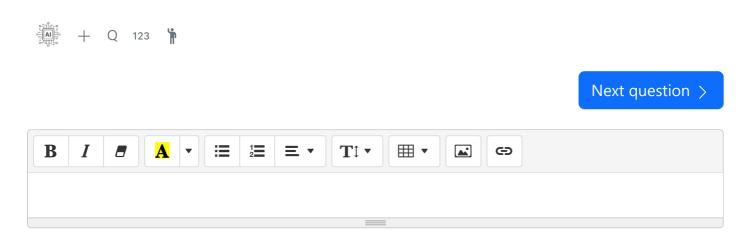
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

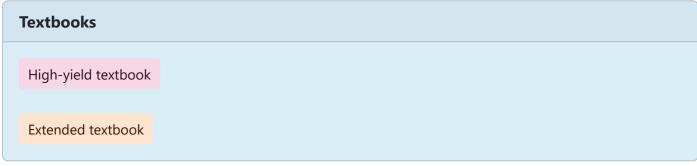
#### Interactions

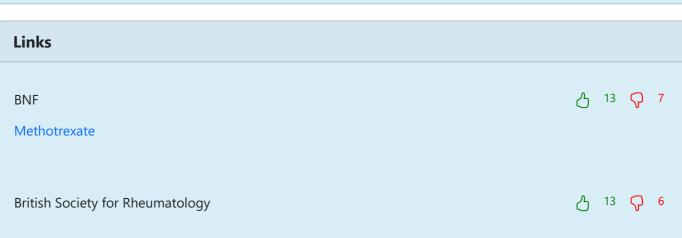
- avoid prescribing <u>trimethoprim</u> or <u>co-trimoxazole</u> concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

#### Methotrexate toxicity

• the treatment of choice is folinic acid







2017 Guideline for the prescription and monitoring of non-biologic disease-modifying anti-rheumatic drugs British Association of Dermatologists △ 12 ♀ 5 2016 Guidelines for the safe and effective prescribing of methotrexate for skin disease Suggest link Report broken link

## Media



Methotrexate - Pharmacology (DMARDs, mechanism of action, side effects)

Armando Hasudungan - YouTube

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Question 48 of 178





A 36-year-old woman presents with recurrent oral ulcerations. She also has widespread joint pain and swelling. More recently she has been losing hair on her scalp.

### Blood results are as follows:

Hb	110 g/L	Male: (135-180) Female: (115 - 160)
Platelets	92 * 10 <sup>9</sup> /L	(150 - 400)
WBC	2.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	135 mmol/L	(135 - 145)
K <sup>+</sup>	4.8 mmol/L	(3.5 - 5.0)
Urea	14.8 mmol/L	(2.0 - 7.0)
Creatinine	162 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)
C3	0.22 g/L	(0.75 - 1.96)
C4	0.04 g/L	(0.14 - 0.54)

What antibody is most likely to be detected?

ANA	
Anti-Smith	
Anti-U1 RNP	
Anti-dsDNA	
Rheumatoid factor	

Submit answer

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Question 48 of 178



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C3	0.22 g/L	(0.75 - 1.96)	
C4	0.04 g/L	(0.14 - 0.54)	

What antibody is most likely to be detected?



Over 99% of patients with SLE are ANA positive, therefore it is a useful rule out test

| Important for me | Less important |

**ANA** is correct. The patient fulfils the diagnostic criteria for systemic lupus erythematosus (SLE). ANA is the most sensitive antibody being positive in >99% of cases.

Anti-Smith is incorrect. Anti-Smith is highly specific for SLE (>99%), but not very sensitive (30%),

**Anti-U1 RNP** is incorrect. This antibody is neither sensitive nor specific for SLE.

Anti-dsDNA is incorrect. Anti-dsDNA is highly specific to SLE (>99%), but less sensitive (70%).

Rheumatoid factor is incorrect. This antibody is neither sensitive nor specific for SLE.



Next question >

# Systemic lupus erythematosus: investigations

#### **Antibodies**

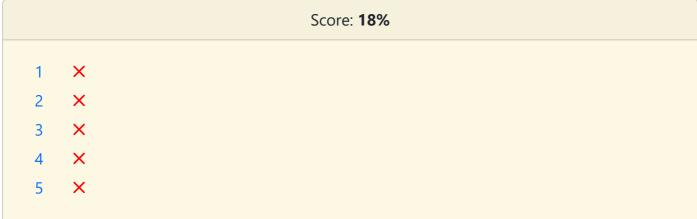
- 99% are ANA positive
  - o this high sensitivity makes it a useful rule out test, but it has low specificity
- 20% are rheumatoid factor positive
- anti-dsDNA: highly specific (> 99%), but less sensitive (70%)
- anti-Smith: highly specific (> 99%), sensitivity (30%)
- also: anti-U1 RNP, SS-A (anti-Ro) and SS-B (anti-La)

#### Monitoring

- inflammatory markers
  - ESR is generally used
  - during active disease the CRP may be normal a raised CRP may indicate underlying infection
- complement levels (C3, C4) are low during active disease (formation of complexes leads to consumption of complement)
- anti-dsDNA titres can be used for disease monitoring (but note not present in all patients)



# **Textbooks** High-yield textbook Extended textbook Links American College of Rheumatology Systemic Lupus Erythematosus diagnostic criteria Suggest link Report broken link Media Systemic Lupus Erythematosus (SLE) - signs and symptoms, pathophysiology, investigations, treatment 占 1 ♀ 0 Armando Hasudungan - YouTube SYSTEMIC LUPUS ERYTHEMATOSUS Systemic lupus erythematosus (SLE) - causes, symptoms, diagnosis & pathology **公** 0 **分** 0 Osmosis - YouTube Report broken media Score: 18%



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A 62-year-old man presents to ED with a six-week history of dry cough and worsening breathlessness. On examination his temperature was 38.4°C and he had fine bibasal crackles on inspiration.

His past medical history was significant for hypertension and psoriatic arthritis for which he had been taking methotrexate for 2 years. Other medications included amlodipine and ramipril. He worked as a farmer and prior to this illness had been quite well.

A chest x-ray showed diffuse ground glass opacities and infiltrates, predominantly affecting the lower lobes.

Hb	142 g/L	Male: (135-180) Female: (115 - 160)
Platelets	161 * 10 <sup>9</sup> /L	(150 - 400)
WBC	8.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Neuts	6.7 * 10 <sup>9</sup> /L	(2.0 - 7.0)
Lymphs	1.4 * 10 <sup>9</sup> /L	(1.0 - 3.5)
Mono	0.6 * 10 <sup>9</sup> /L	(0.2 - 0.8)
Eosin	0.8 * 10 <sup>9</sup> /L	(0.0 - 0.4)

What is the likely diagnosis?

	Hypersensitivity pneumonitis (Farmer's lung)	
0	Interstitial lung disease	
0	Methotrexate induced pneumonitis	
0	Pneumocystis jiroveci pneumonia	
	Viral pneumonitis	

Submit answer

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Question 49 of 178



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Mono	0.6 * 10 <sup>9</sup> /L	(0.2 - 0.8)
Eosin	0.8 * 10 <sup>9</sup> /L	(0.0 - 0.4)

What is the likely diagnosis?

Hypersensitivity pneumonitis (Farmer's lung)	22%
Interstitial lung disease	7%
Methotrexate induced pneumonitis	68%
Pneumocystis jiroveci pneumonia	3%
Viral pneumonitis	1%

Methotrexate may cause pneumonitis - typically presents with cough, dyspnoea and fever

| Important for me | Less important |

This patient has presented with cough, dyspnoea and fever with bibasal fine crackles while on methotrexate. This, combined with the presence of infiltrates on chest x-ray, should raise the

suspicion of methotrexate pneumonitis.

**Hypersensitivity pneumonitis (Farmer's lung)** would not be an unreasonable differential diagnosis. However, in acute farmer's lung patients would however present with a short history of fevers, chills, malaise, headache and myalgia alongside breathlessness and dry cough.

**Interstitial lung disease** would present with a longer history of breathlessness and without fever or infiltrates on chest x-ray. They may also have finger clubbing on examination.

**Pneumocystis jiroveci pneumonia** would be unusual without significant immunocompromise, such as infection with untreated HIV. It would however present with fever, exertional dyspnoea and dry cough and fine interstitial changes on chest x-ray. Given there is no history of significant immunocompromise, this is less likely than methotrexate-induced pneumonitis.

**Viral pneumonitis** such as COVID-19 infection is also not an unreasonable differential given the chest x-ray changes but it would be unusual to have a 6-week history and there is no significant upper respiratory tract symptoms or systemic features of rigors or myalgia which you might expect with a viral illness.



Next question >

# Methotrexate ★

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

#### Indications

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

#### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - o typically develops within a year of starting treatment, either acutely or subacutely

- o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

#### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

#### Prescribing methotrexate

- methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

#### Interactions

- avoid prescribing <u>trimethoprim</u> or <u>co-trimoxazole</u> concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

#### Methotrexate toxicity

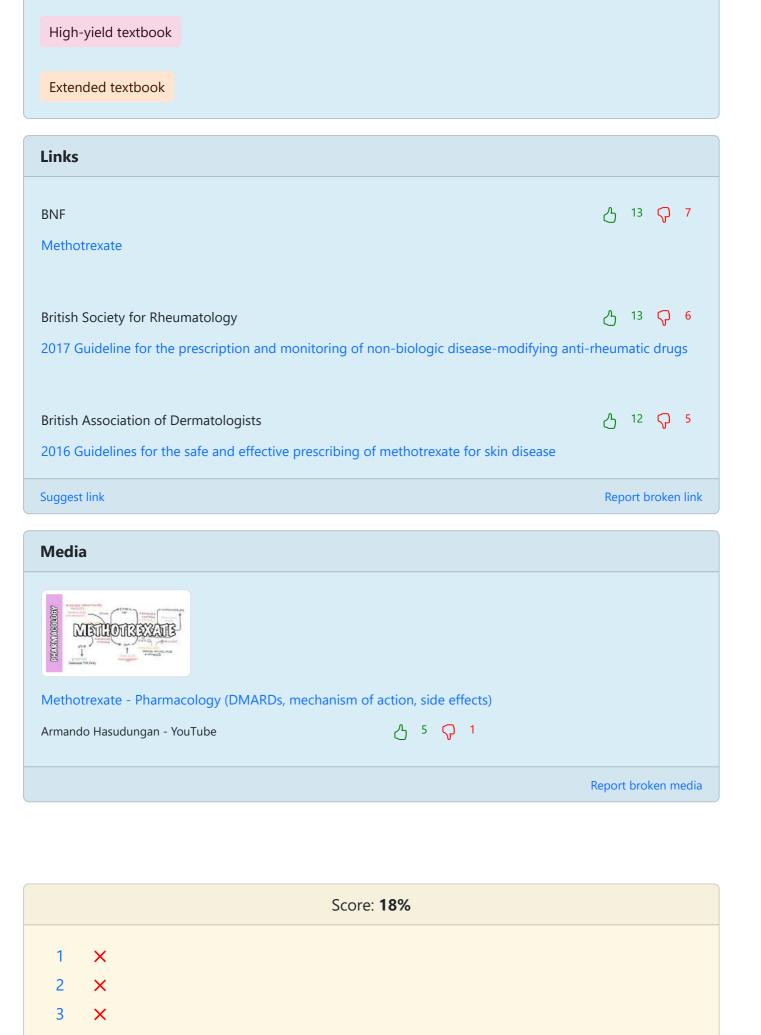
the treatment of choice is folinic acid



Next question >



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Question 50 of 178





A 54-year-old woman with a background of rheumatoid arthritis is being managed with a weekly dose of 15mg of methotrexate. She has presented for regular review and her blood tests indicate that she has had a significant fall in her cell counts. It is thought that she has methotrexate induced bone marrow failure.

Hb	110 g/l	
Platelets	135* 10 <sup>9</sup> /I	
WBC	3 * 10 <sup>9</sup> /l	
Neutrophils	1.9*10 <sup>9</sup> /l	

What is the best management option?

Folinic acid	
Ferrous sulphate	
Folic acid	
Iron dextran	
Palifermin	

Submit answer

Reference ranges  $\vee$ 

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Question 50 of 178



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Hb	110 g/l	
Platelets	135* 10 <sup>9</sup> /l	
WBC	3 * 10 <sup>9</sup> /l	
Neutrophils	1.9*10 <sup>9</sup> /I	

What is the best management option?



Folinic acid is the treatment of choice for methotrexate toxicity

Important for me Less important



The recommended treatment for myelosuppression secondary to her methotrexate therapy is with folinic acid rescue therapy.

Palifermin is used for oral mucositis associated with methotrexate treatment for haematological malignancies. Folic acid is used to prevent methotrexate associated side effects. Ferrous sulphate and iron dextran are not indicated in this case as they are used for iron deficiency anaemia.



### Methotrexate ★

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

### Indications

- inflammatory arthritis, especially rheumatoid arthritis
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- some chemotherapy acute lymphoblastic leukaemia

### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - o typically develops within a year of starting treatment, either acutely or subacutely
  - o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
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### Interactions

- avoid prescribing trimethoprim or co-trimoxazole concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

### Methotrexate toxicity

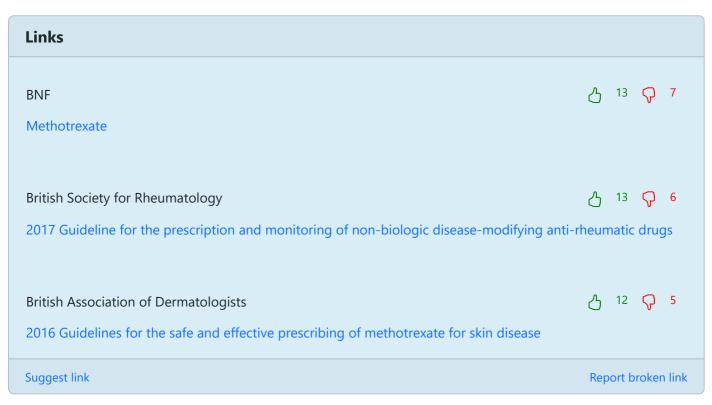
• the treatment of choice is folinic acid



Next question >







### Media



Methotrexate - Pharmacology (DMARDs, mechanism of action, side effects)

Armando Hasudungan - YouTube





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### Question 51 of 178





A 67-year-old Caucasian man presents with progressive deafness and difficulty chewing. He also states that his father and paternal uncles suffered from similar symptoms that required medication. On examination, it is noted that there is frontal bossing. Further investigations find an elevated alkaline phosphatase and a serum calcium at the upper end of the normal range. His other investigations are normal. What is the best first-line treatment for this man?

Calcium supplementation	
Bisphosphonates	
Calcium and Vitamin D supplementation	
Calcitonin	
Surgery	

Submit answer

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Question 51 of 178



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Paget's disease of the bone is treated with bisphosphonates

Important for me Less important

This gentleman has findings consistent with Paget's disease of the bone given his clinical symptoms, ethnic background, family history and biochemical results. The NICE guidelines recommend bisphosphonates as first-line treatment in symptomatic patients. In asymptomatic patients, watchful waiting may be sufficient initially.

Supplementation is insufficient in this patient given that he is symptomatic.

Surgery may be required if there are complications such as fractures or severe osteoarthritis but not at this stage.



Next question >

# Paget's disease of the bone \*

Paget's disease is a disease of increased but uncontrolled bone turnover. It is thought to be primarily a disorder of osteoclasts, with excessive osteoclastic resorption followed by increased

osteoblastic activity. Paget's disease is common (UK prevalence 5%) but symptomatic in only 1 in 20 patients. The skull, spine/pelvis, and long bones of the lower extremities are most commonly affected.

### Predisposing factors

- increasing age
- male sex
- northern latitude
- family history

### Clinical features - only 5% of patients are symptomatic

- the stereotypical presentation is an older male with bone pain and an isolated raised ALP
- bone pain (e.g. pelvis, lumbar spine, femur)
- classical, untreated features: bowing of tibia, bossing of skull

### Investigations

- bloods
  - raised alkaline phosphatase (ALP)
  - calcium and phosphate are typically normal. Hypercalcaemia may occasionally occur with prolonged immobilisation
- other markers of bone turnover include
  - o procollagen type I N-terminal propeptide (PINP)
  - serum C-telopeptide (CTx)
  - o urinary N-telopeptide (NTx)
  - urinary hydroxyproline
- x-rays
  - o osteolysis in early disease → mixed lytic/sclerotic lesions later
  - o skull x-ray: thickened vault, osteoporosis circumscripta
- bone scintigraphy
  - increased uptake is seen focally at the sites of active bone lesions

### Management

- indications for treatment include
  - o bone pain
  - skull or long bone deformity
  - fracture
  - o periarticular Paget's
- bisphosphonate (either oral risedronate or IV zoledronate)
- calcitonin is less commonly used now

### Complications

- deafness (cranial nerve entrapment)
- bone sarcoma (1% if affected for > 10 years)
- fractures

- skull thickening
- high-output cardiac failure





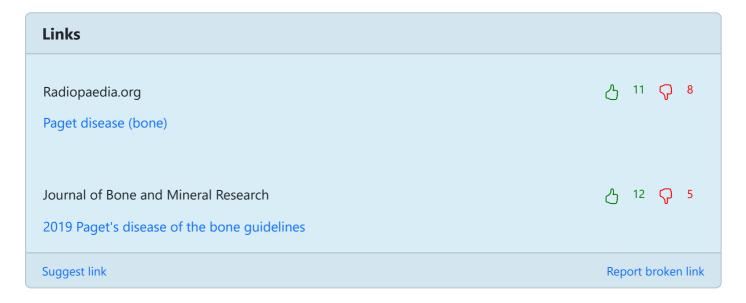




Next question >



# Textbooks High-yield textbook Extended textbook



# Media



### Paget's Disease of the bone

Armando Hasudungan - YouTube  $\bigcirc$  0  $\bigcirc$  0









### Paget's disease of the bone

Osmosis - YouTube





Report broken media

### Score: **14%**

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Question 52 of 178





A 28-year-old woman is referred to rheumatology by her general practitioner complaining of a one-year history of painful hands. She describes daily episodes of her hands changing colour and becoming painful, especially in cold weather. Firstly they go white, then blue and then red, which is followed by a resolution of the symptoms.

She denies any history of rash, chest pain, weakness, mouth ulcers, skin thickening, blood clots, fever or joint swelling. She has no significant past medical history and is on no regular medications. She does not smoke, drink alcohol or take any recreational drugs. There is no relevant family history. She works as a teacher. Despite wearing gloves regularly and trying to avoid the freezer aisle in the supermarket, her symptoms have persisted.

On clinical examination, her hands demonstrated a blue-white discolouration and were cool to touch. There was no evidence of digital ulceration or scarring. Her skin was normal and there was no synovitis. There was no clinical evidence of interstitial lung disease or pulmonary hypertension. Her pulses were present in her upper limbs. There was no proximal muscle weakness.

### Blood test results:

Antinuclear antibody	negative
Rheumatoid factor	negative
ANCA	negative
Lupus anticoagulant	negative
Anticardiolipin antibody	negative
HIV	negative
Hepatitis B surface antigen	negative
Hepatitis C antibody	negative
Hepatitis B core antibody	negative

Given the likely diagnosis, what is the appropriate initial pharmacological management?

Aspirin	
Intravenous iloprost	
Nifedipine	

Prednisolone	
Sildenafil	

### Submit answer

Reference ranges ✓

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Question 52 of 178



 $\Box$ 



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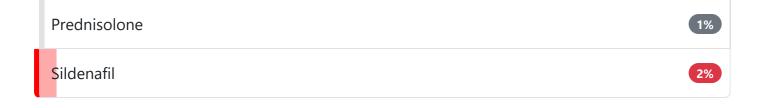
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Anticardiolipin antibody	negative
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Hepatitis B surface antigen	negative
Hepatitis C antibody	negative
Hepatitis B core antibody	negative

Given the likely diagnosis, what is the appropriate initial pharmacological management?





Nifedipine is a pharmacological option for Raynaud's phenomenon

Important for me Less important



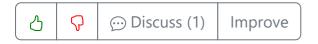
Nifedipine is the correct answer. This is the first-line treatment in those with Raynaud's phenomenon where conservative measures have failed. This patient is likely to have primary Raynaud's phenomenon as there is no clinical or serological evidence of a predisposing underlying condition.

Aspirin is not the correct answer as it is not typically indicated in the symptomatic management of Raynaud's phenomenon. If the patient had a condition like anti-phospholipid syndrome, resulting in secondary Raynaud's phenomenon, then there might be a place for aspirin in management of the underlying condition.

Intravenous iloprost is incorrect. This is reserved for severe cases of Raynaud's phenomenon that have not responded to first-line treatment or those cases characterized by digital ischaemia.

Prednisolone is incorrect. This is used to induce remission in many autoimmune and connective tissue diseases. However, there is no suggestion from the clinical vignette that this patient has a serious underlying condition.

Sildenafil is incorrect. This can be used to treat Raynaud's phenomenon but would not be the first-line choice in primary Raynaud's.



Next question >

## Raynaud's phenomenon \*

Raynaud's phenomenon is characterised by an exaggerated vasoconstrictive response of the digital arteries and cutaneous arteriole to the cold or emotional stress. It may be primary (Raynaud's disease) or secondary (Raynaud's phenomenon).

Raynaud's disease typically presents in young women (e.g. 30 years old) with bilateral symptoms.

Secondary causes of Raynaud's phenomenon

- connective tissue disorders
  - scleroderma (most common)
  - rheumatoid arthritis
  - o systemic lupus erythematosus
- leukaemia
- type I cryoglobulinaemia, cold agglutinins
- use of vibrating tools
- drugs: oral contraceptive pill, ergot
- cervical rib

### Factors suggesting underlying connective tissue disease

- onset after 40 years
- unilateral symptoms
- rashes
- presence of autoantibodies
- features which may suggest rheumatoid arthritis or SLE, for example arthritis or recurrent miscarriages
- digital ulcers, calcinosis
- very rarely: chilblains

### Management

- all patients with suspected secondary Raynaud's phenomenon should be referred to secondary care
- first-line: calcium channel blockers e.g. nifedipine
- IV prostacyclin (epoprostenol) infusions: effects may last several weeks/months



Next question >



### **Textbooks**

High-yield textbook

Extended textbook

Links	
Arthritis Research Council (ARC)  ARC Raynaud's Patient Info	<u></u> 5
Suggest link	Report broken link

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Question 53 of 178





A 65-year-old male presents to the emergency department with 5 days of haemoptysis, fever and joint pains. He has a past medical history of hypertension and chronic sinusitis. He takes amlodipine. He smokes 10 cigarettes daily and drinks wine 1-2x/week. He has recently returned from a holiday in Goa.

His observations are heart rate 101 beats per minute, blood pressure 167/94 mmHg, oxygen saturations 94%, respiratory rate 21/minute and temperature 37.9°C.

Chest auscultation reveals scattered crackles and decreased air entry at the right base. There is mild tenderness and swelling at the wrists bilaterally. Abdominal examination is normal. There is no peripheral oedema.

### Urinalysis:

Leucocytes	-ve
Nitrites	-ve
Protein	++
Blood	+++
Glucose	-ve

### Blood tests:

Hb	124 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10 <sup>9</sup> /L	(150 - 400)
WBC	8.7 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Neuts	5.2 * 10 <sup>9</sup> /L	(2.0 - 7.0)
Lymphs	3.2 * 10 <sup>9</sup> /L	(1.0 - 3.5)
Mono	0.2 * 10 <sup>9</sup> /L	(0.2 - 0.8)
Eosin	0.1 * 10 <sup>9</sup> /L	(0.0 - 0.4)
Na <sup>+</sup>	129 mmol/L	(135 - 145)
K <sup>+</sup>	4.8 mmol/L	(3.5 - 5.0)
Bicarbonate	21 mmol/L	(22 - 29)
Urea	9.2 mmol/L	(2.0 - 7.0)
Creatinine	241 µmol/L	(55 - 120)

CRP	180 mg/dL	(<5)
Creatinine kinase	100 U/L	(38-174)

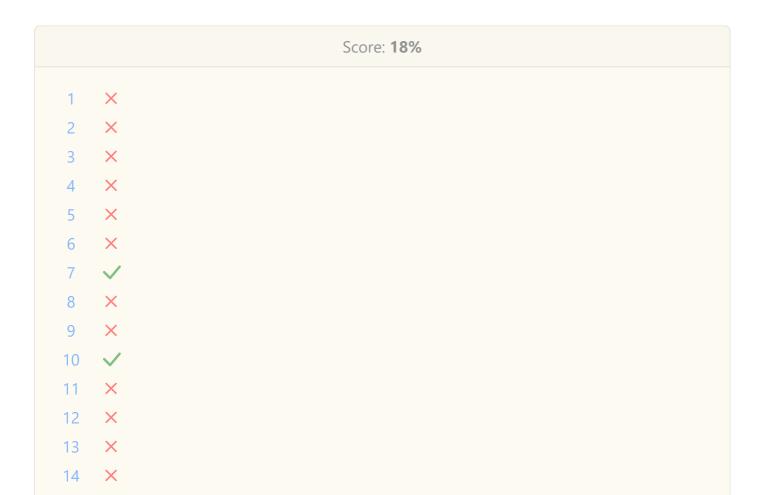
A chest x-ray reveals patchy airspace opacification in the lung fields bilaterally.

What is the most likely diagnosis?

	Churg-Strauss syndrome	
0	Dermatomyositis	×
0	Goodpasture's syndrome	×
0	Granulomatosis with polyangiitis	×
	Systemic lupus erythematosus	

Submit answer

Reference ranges  $\checkmark$ 



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Question 53 of 178



 $\Box$ 



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Creatinine	241 µmol/L	(55 - 120)

CRP	180 mg/dL	(<5)
Creatinine kinase	100 U/L	(38-174)

A chest x-ray reveals patchy airspace opacification in the lung fields bilaterally.

What is the most likely diagnosis?



Renal impairment, respiratory symptoms, joint pain, systemic features → consider ANCA associated vasculitis

Important for me Less important

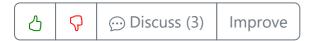
Granulomatosis with polyangiitis is the correct answer. This man presents with haemoptysis, fever and polyarthralgia and has potential evidence of glomerulonephritis (haematuria, proteinuria) with an acute kidney injury and likely pulmonary haemorrhage. The background of sinusitis makes this the most likely diagnosis from the options listed.

Goodpasture's syndrome is incorrect. This can cause a pulmonary-renal syndrome but joint and sinus involvement are less likely.

Churg-Strauss syndrome is incorrect. This can cause a pulmonary-renal syndrome but the eosinophil count is normal, essentially excluding this diagnosis.

Systemic lupus erythematosus is incorrect. This is a possible cause of pulmonary-renal syndrome, however, CRP tends to decrease in active SLE (it is a marker of disease activity), pulmonary haemorrhage is relatively rare in this condition and it is unlikely to present with lupus at age 65 in a male patient.

Dermatomyositis is incorrect. There is no rash or muscle weakness, making this an unlikely cause of pulmonary haemorrhage in this case.



## ANCA associated vasculitis \*

Anti-neutrophil cytoplasmic antibodies (ANCA) are important as they are associated with a number of small-vessel vasculitides, including:

- granulomatosis with polyangiitis
- eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)
- microscopic polyangiitis

ANCA associated vasculitis is more common with increasing age. Whilst each condition has its own distinct features, there are a number of common findings:

- renal impairment
  - caused by immune complex glomerulonephritis → raised creatinine, haematuria and proteinuria
- respiratory symptoms
  - dyspnoea
  - o haemoptysiis
- systemic symptoms
  - o fatique
  - weight loss
  - o fever
- vasculitic rash: present only in a minority of patients
- ear, nose and throat symptoms
  - o sinusitis

General approach to first-line investigations:

- urinalysis for haematuria and proteinuria
- bloods:
  - urea and creatinine for renal impairment
  - o full blood count: normocytic anaemia and thrombocytosis may be seen
  - CRP: raised
  - ANCA testing (see below)
- chest x-ray: nodular, fibrotic or infiltrative lesions may be seen

## **ANCA** types

There are two main types of ANCA - cytoplasmic (cANCA) and perinuclear (pANCA). There is considerable overlap between which antibodies are found in which condition, but as a rule of thumb:

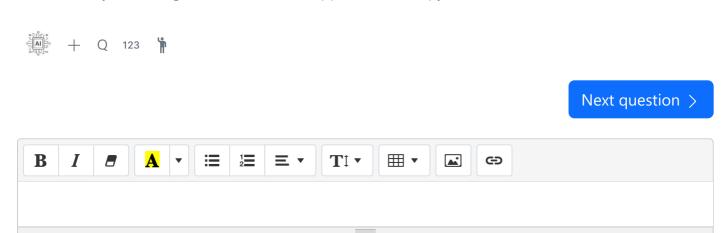
- cANCA granulomatosis with polyangiitis
- pANCA eosinophilic granulomatosis with polyangiitis + others (see below)

	cANCA	pANCA
Target	Serine proteinase 3 (PR3)	myeloperoxidase (MPO)
Granulomatosis with polyangiitis	90%	25%
Eosinophilic granulomatosis with polyangiitis	Low	50%
Microscopic polyangiitis	40%	75%
Other associated conditions		Ulcerative colitis (70%) Primary sclerosing cholangitis (70%) Anti-GBM disease (25%) Crohn's disease (20%)
Use for monitoring	Some correlation between cANCA levels and disease activity	Cannot use level of pANCA to monitor disease activity

# General approach to management

Once suspected, ANCA associated vasculitis should be managed by specialist teams (e.g. renal, rheumatology, respiratory) to allow an exact diagnosis to be made. Kidney or lung biopsies may be taken to aid the diagnosis.

The mainstay of management is immunosuppressive therapy.



# Textbooks

High-yield textbook

Extended textbook

### Media



ANCA (Anti-Neutrophil Cytoplasmic Antibody) Associated Vasculitis - Causes, Pathophysiology, Types

Armando Hasudungan - YouTube



Report broken media

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Question 54 of 178





A 77-year-old woman presents to the emergency department with a fall. She tripped on a wet surface in her kitchen while cooking lunch and sustained an injury to her wrist. She has a past medical history of hypertension and takes ramipril. She does not smoke or drink alcohol. She is independent and lives with her husband. She paints in her spare time.

Examination reveals tenderness and swelling at the right wrist and is otherwise unremarkable.

Plain radiography of the right wrist demonstrates a Colles fracture.

### Blood tests:

Hb	120 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10 <sup>9</sup> /L	(150 - 400)
WBC	4.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	136 mmol/L	(135 - 145)
K <sup>+</sup>	4.2 mmol/L	(3.5 - 5.0)
Urea	4.4 mmol/L	(2.0 - 7.0)
Creatinine	89 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
Calcium	2.24 mmol/L	(2.20-2.6)
Vitamin D	75 nmol/L	(>50)
ALP	180 IU/L	(44-147)

She is managed conservatively without the need for an operation.

From the list below, what is the most appropriate next step in management?

Alendronic acid	
Bone density (DEXA) scan	
Denosumab	
Teriparatide	
IV Zoledronic acid	

# Submit answer

Reference ranges  $\checkmark$ 

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Question 54 of 178







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She is managed conservatively without the need for an operation.

From the list below, what is the most appropriate next step in management?

Alendronic acid	70%
Bone density (DEXA) scan	24%
Denosumab	2%
Teriparatide	0%
IV Zoledronic acid	4%

Start alendronate in patients >= 75 years following a fragility fracture, without waiting for a **DEXA** scan

Important for me Less important

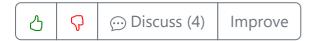
Alendronic acid is the correct answer. In patients >= 75 years of age who have sustained a fragility fracture, bone protection can be started without the need for a DEXA scan. In the absence of contraindications, first-line management is typically an oral bisphosphonate such as alendronate.

Bone density (DEXA) scan is incorrect. Treatment can be commenced without this investigation given the clinical context.

IV Zoledronic acid is incorrect. IV bisphosphonates are a second line treatment for osteoporosis, typically used if there is an intolerance or contraindication to oral bisphosphonates.

Denosumab is incorrect. This is a second line treatment for osteoporosis, typically used if oral bisphosphonates are not tolerated or contraindicated. It is also a more appropriate choice if the creatinine clearance is < 30ml/minute.

Teriparatide is incorrect. This has strict criteria for usage and is typically only trialled after first-line and second-line treatments for osteoporosis have been used.



Next question >

## Osteoporosis: Assessing patients following a fragility fracture



The management of patients following a fragility fracture depends on age.

## Patients >= 75 years of age

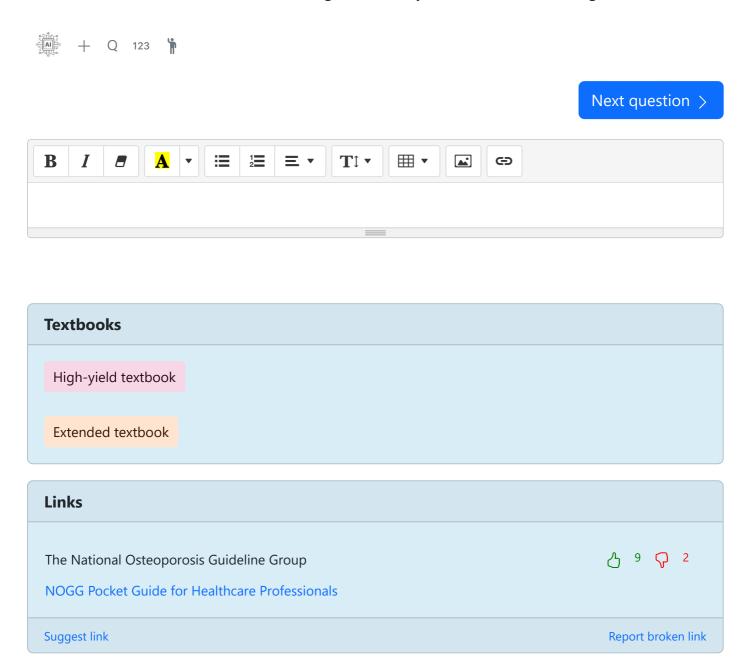
Patients who've had a fragility fracture and are >= 75 years of age are presumed to have underlying osteoporosis and should be started on first-line therapy (an oral bisphosphonate), without the need for a DEXA scan.

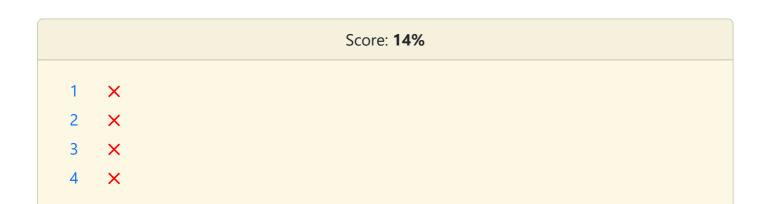
It should be noted that the 2014 NOGG guidelines have a different threshold, suggesting treatment is started in all women over the age of 50 years who've had a fragility fracture -'although BMD measurement may sometimes be appropriate, particularly in younger postmenopausal women.'

## Patients < 75 years of age

If a patient is under the age of 75 years a DEXA scan should be arranged. These results can then be entered into a FRAX assessment (along with the fact that they've had a fracture) to determine the patients ongoing fracture risk.

For example, a 79-year-old woman falls over on to an outstretched hand and sustains a Colles' fracture (fracture of the distal radius). Given her age she is presumed to have osteoporosis and therefore started on oral alendronate 70mg once weekly. No DEXA scan is arranged.





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Question 55 of 178





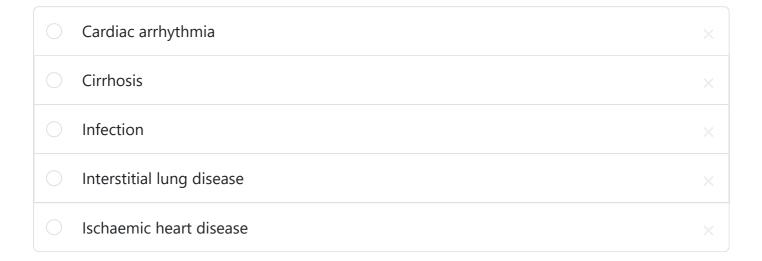
A 45-year-old woman is referred to rheumatology with a new-onset Raynaud's phenomenon. She has no past medical history.

On examination, her fingers are cool and pale. The skin is hard and thick from the hands to the mid-forearms. There are small, dilated blood vessels on her hands and on her face.

### Blood test:

anti Scl-70 positive

What is the most common cause of death in this condition?



Submit answer

Reference ranges ∨

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Question 55 of 178







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On examination, her fingers are cool and pale. The skin is hard and thick from the hands to the mid-forearms. There are small, dilated blood vessels on her hands and on her face.

Blood test:

tive

What is the most common cause of death in this condition?



The most common cause of death in systemic sclerosis is respiratory involvement: **interstitial lung disease** and **pulmonary arterial hypertension** 

Important for me Less important



**Interstitial lung disease** is correct. The patient has systemic sclerosis as evidenced by Raynaud's phenomenon, skin hardening, telangiectasias (small, dilated blood vessels near the surface of the skin) and positivity for Scl-70 antibody. The most common causes of mortality in systemic sclerosis generally are interstitial lung disease and pulmonary hypertension. Scl-70 antibody is associated with the development of interstitial lung disease.

**Cardiac arrhythmia** is incorrect. Arrhythmias may result from conduction system fibrosis in this condition. However, conduction disease is felt to be uncommon overall in this condition and is often asymptomatic and therefore not a significant cause of mortality.

Cirrhosis is incorrect. Primary biliary cirrhosis and autoimmune hepatitis can occur in systemic

sclerosis. However, they are relatively rare complications and not associated with the same mortality as ILD in this condition.

**Infection** is not the right answer. Patients with systemic sclerosis are at higher risk of infectious complications due to structural damage from the disease and the use of immunosuppressive treatment. However, interstitial lung disease still represents a more common cause of mortality in this condition.

**Ischaemic heart disease** is incorrect. There is an increased rate of ischaemic heart disease in patients with systemic sclerosis compared to the general population. However, there is a less dramatic increase when compared with other autoimmune diseases like systemic lupus erythematosus and rheumatoid arthritis. It is a less common cause of death than ILD.



Next question >

# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - patients with renal disease should be started on an ACE inhibitor
- poor prognosis

Scleroderma (without internal organ involvement)

• tightening and fibrosis of skin

• may be manifest as plaques (morphoea) or linear



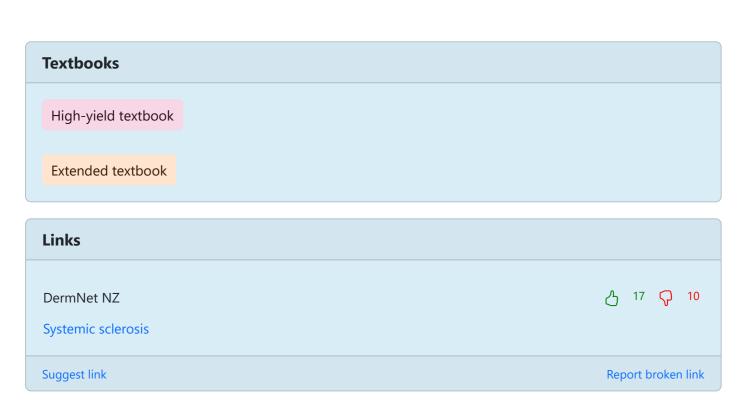


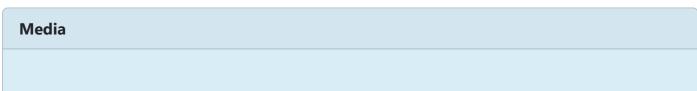


### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis









### Scleroderma

Townsend Teaching - YouTube 4 Q 0









### Scleroderma

Osmosis - YouTube











## Systemic Sclerosis and Scleroderma

Zero to Finals - YouTube







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A 31-year-old woman with presents with three days of right sided loin pain with two episodes of blood in her urine. She feels unwell and lethargic but denies any fevers or urinary dysuria.

She is usually well and has had two urinary tract infections previously. She is being seen by the anticoagulants clinic due to several miscarriages and is due for review next week. Her maternal aunt has previous renal calculi. She takes no regular medication at present.

On examination, she is tender in the right loin only. Her blood pressure is 180/105 mmHg, heart rate 85/min, respiratory rate 22/min and temperature 37.0°C.

Na <sup>+</sup>	138 mmol/l
K <sup>+</sup>	4.2 mmol/l
Urea	5.6 mmol/l
Creatinine	87 µmol/l
Anti-cardiolipin antibodies	positive

Urine dip	blood +++, Leu -
Ultrasound KUB	no hydronephrosis or renal lesion seen

What is the likely diagnosis?

Renal calculus	
Urinary tract infection	
Renal cell carcinoma	
Renal vein thrombosis	
Tubulointerstitial nephritis	

Submit answer

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Question 56 of 178



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Urea	5.6 mmol/l
Creatinine	87 µmol/l
Anti-cardiolipin antibodies	positive

Urine dip	blood +++, Leu -
Ultrasound KUB	no hydronephrosis or renal lesion seen

What is the likely diagnosis?



Loin pain + haematuria in antiphospholipid syndrome -> renal vein thrombus

Important for me Less important



Patients with antiphospholipid syndrome (APS) are at risk of thromboses and loin pain with haematuria in hypercoagulable states is most likely a renal thrombus. Although not formally diagnosed, she has positive antibodies and multiple miscarriages diagnostic of APS. She could have a renal calculus though this less often has frank haematuria and there are no risk factors for it. A UTI is unlikely given the lack of lower urinary tract symptoms. Tubulointerstitial nephritis often has a rash and the urine dip has leucocytes and eosinophilic casts on microscopy.



Next question >

# Antiphospholipid syndrome \*

Antiphospholipid syndrome is an acquired disorder characterised by a predisposition to both venous and arterial thromboses, recurrent fetal loss and thrombocytopenia. It may occur as a primary disorder or secondary to other conditions, most commonly systemic lupus erythematosus (SLE). Around 30% of patients with SLE have positive antiphospholipid antibodies.

A key point for the exam is to appreciate that antiphospholipid syndrome causes a paradoxical rise in the APTT. This is due to an ex-vivo reaction of the lupus anticoagulant autoantibodies with phospholipids involved in the coagulation cascade.

#### Associations other than SLE

- other autoimmune disorders
- lymphoproliferative disorders
- phenothiazines (rare)

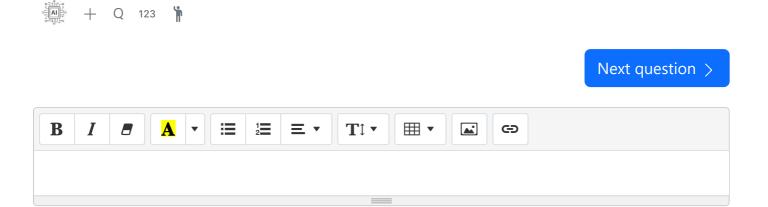
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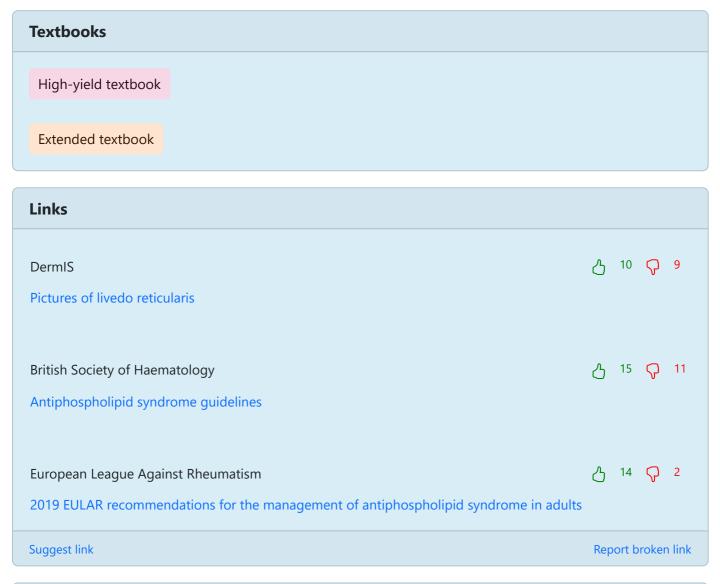
- venous/arterial thrombosis
- recurrent miscarriages
- livedo reticularis
- other features: pre-eclampsia, pulmonary hypertension

### Investigations

- antibodies
  - anticardiolipin antibodies
  - o anti-beta2 glycoprotein I (anti-beta2GPI) antibodies
  - lupus anticoagulant
- thrombocytopenia
- prolonged APTT

- primary thromboprophylaxis
  - o low-dose aspirin
- secondary thromboprophylaxis
  - o initial venous thromboembolic events: lifelong warfarin with a target INR of 2-3
  - recurrent venous thromboembolic events: lifelong warfarin; if occurred whilst taking warfarin then consider adding low-dose aspirin, increase target INR to 3-4
  - o arterial thrombosis should be treated with lifelong warfarin with target INR 2-3









### Antiphospholipid syndrome

Osmosis - YouTube







Clues to the Diagnosis of Antiphospholipid Syndrome (Hughes' Syndrome)

Prof Graham Hughes - YouTube





Report broken media

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### Ouestion 57 of 178





A 67-year-old gentleman suffering from Rheumatoid Arthritis is admitted to hospital with short history of weakness and numbness in the left side of his face. The doctor that assesses him in the emergency department refers him urgently to the stroke unit where a CT head took place that is unremarkable.

The symptoms did not resolve, and as such he was loaded and treated for potential cerebrovascular accident. An MRI was booked alongside a carotid Doppler both which were unremarkable.

You are asked to see him on-call by the nurse informing you that he has been pyrexial for a few days now despite analgesia with normal blood cultures. His urine dip however has consistently had proteinuria. He tells you that before his admission he was troubled by foot drop of the right foot, which has now resolved.

You examine this patient and beside the weakness and loss of sensation associated with the facial and trigeminal nerves, it is otherwise unremarkable. There are no added heart sound on chest auscultation, and no splinter haemorrhages or clubbing. Breathing sounds are vesicular. He is otherwise neurologically intact. No rashes are noted.

### You review his bloods

Hb	105 g/l	Na <sup>+</sup>	137 mmol/l
Platelets	468 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.5 mmol/l
WBC	10.2 * 10 <sup>9</sup> /I	Urea	13.4 mmol/l
Neuts	7.2 * 10 <sup>9</sup> /l	Creatinine	245 µmol/l
Lymphs	2.1 * 10 <sup>9</sup> /l	CRP	265 mg/l
Eosin	0.6 * 10 <sup>9</sup> /I	ESR	88 mm/h
ANA	1:640	ANCA	positive cANCA

What is the most appropriate investigation to verify this patient's cause of symptoms?

Renal biopsy	
CT thorax/abdomen/pelvis	
Virology screen	
Transthoracic Echocardiography	

# Submit answer

Reference ranges  $\checkmark$ 

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Ouestion 57 of 178



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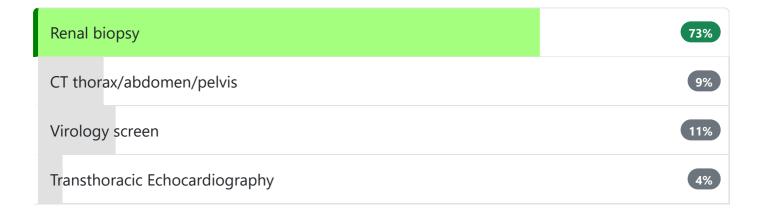
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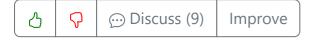
What is the most appropriate investigation to verify this patient's cause of symptoms?



Biopsy (renal, nasal, skin) can be useful in the diagnosis of ANCA associated vasculitis (e.g. glomerulonephritis with polyangiitis)

Important for me Less important

This patient presents with a history of potential mononeuritis multiplex. The causes of mononeuritis multiplex are numerous; however this history and the blood investigation findings (including raised ESR, CRP, raised cANCA, reduced eGFR and proteinuria) point to the potential of an ANCA associated vasculitis; in this case Granulomatosis with polyangiitis The gold standard of investigation in such cases is biopsy, with renal biopsy being the most appropriate option given the involvement of kidneys in this patient. It is understandable that a doctor could be tricked in identifying an infectious cause / malignant cause (paraneoplastic syndrome) however given the negative blood cultures, lack of examination findings associated with infection and strong indication of a systemic inflammatory response, a renal biopsy could be appropriate.



Next question >

# ANCA associated vasculitis \*

Anti-neutrophil cytoplasmic antibodies (ANCA) are important as they are associated with a number of small-vessel vasculitides, including:

- granulomatosis with polyangiitis
- eosinophilic granulomatosis with polyangiitis (Churg-Strauss syndrome)
- microscopic polyangiitis

ANCA associated vasculitis is more common with increasing age. Whilst each condition has its own distinct features, there are a number of common findings:

- renal impairment
  - caused by immune complex glomerulonephritis → raised creatinine, haematuria and proteinuria
- respiratory symptoms
  - o dyspnoea
  - haemoptysiis
- systemic symptoms
  - fatigue
  - weight loss
  - o fever
- vasculitic rash: present only in a minority of patients

- ear, nose and throat symptoms
  - sinusitis

General approach to first-line investigations:

- urinalysis for haematuria and proteinuria
- bloods:
  - urea and creatinine for renal impairment
  - o full blood count: normocytic anaemia and thrombocytosis may be seen
  - o CRP: raised
  - ANCA testing (see below)
- chest x-ray: nodular, fibrotic or infiltrative lesions may be seen

# **ANCA** types

There are two main types of ANCA - cytoplasmic (cANCA) and perinuclear (pANCA). There is considerable overlap between which antibodies are found in which condition, but as a rule of thumb:

- cANCA granulomatosis with polyangiitis
- pANCA eosinophilic granulomatosis with polyangiitis + others (see below)

	cANCA	pANCA
Target	Serine proteinase 3 (PR3)	myeloperoxidase (MPO)
Granulomatosis with polyangiitis	90%	25%
Eosinophilic granulomatosis with polyangiitis	Low	50%
Microscopic polyangiitis	40%	75%
Other associated conditions		Ulcerative colitis (70%) Primary sclerosing cholangitis (70%) Anti-GBM disease (25%) Crohn's disease (20%)
Use for monitoring	Some correlation between cANCA levels and disease activity	Cannot use level of pANCA to monitor disease activity

# General approach to management

Once suspected, ANCA associated vasculitis should be managed by specialist teams (e.g. renal, rheumatology, respiratory) to allow an exact diagnosis to be made. Kidney or lung biopsies may be taken to aid the diagnosis.

The mainstay of management is immunosuppressive therapy.



Next question >

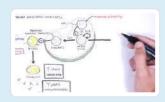




High-yield textbook

Extended textbook

## Media



ANCA (Anti-Neutrophil Cytoplasmic Antibody) Associated Vasculitis - Causes, Pathophysiology, Types

Armando Hasudungan - YouTube



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### Question 58 of 178





You see a 29-year-old patient with a swollen left knee. It has been getting worse for a number of months and is now affecting his ability to work as a postman. The pain and stiffness are worse overnight and in the morning and gets better throughout the day. He denies any trauma or fever. He also has some pain and stiffness in his fingers which is also worse in the morning. His joints are worse after rest.

He is normally fit and well and denies any previous joint problems or psoriasis. However, his father has a history of psoriasis. He says there is no family history of joint problems to his knowledge.

On examination, his knee is red, warm and tender to palpate. There is a moderate knee effusion and flexion is limited due to pain. His distal interphalangeal joints (DIPs) are tender and slightly swollen.

There are no skin rashes. Although his knee is painful the patient looks well and has a heart rate of 80 beats per minute and his temperature is 36.8°C.

Blood tests reveal a raised CRP at 85 but a negative rheumatoid factor.

What is the most likely diagnosis?

Juvenile idiopathic arthritis	
Seronegative rheumatoid arthritis	
Psoriatic arthritis	
Septic arthritis	
Rheumatoid arthritis	

Submit answer

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 $\Box$ 



You see a 29-year-old patient with a swollen left knee. It has been getting worse for a number of months and is now affecting his ability to work as a postman. The pain and stiffness are worse overnight and in the morning and gets better throughout the day. He denies any trauma or fever. He also has some pain and stiffness in his fingers which is also worse in the morning. His joints are worse after rest.

He is normally fit and well and denies any previous joint problems or psoriasis. However, his father has a history of psoriasis. He says there is no family history of joint problems to his knowledge.

On examination, his knee is red, warm and tender to palpate. There is a moderate knee effusion and flexion is limited due to pain. His distal interphalangeal joints (DIPs) are tender and slightly swollen.

There are no skin rashes. Although his knee is painful the patient looks well and has a heart rate of 80 beats per minute and his temperature is 36.8°C.

Blood tests reveal a raised CRP at 85 but a negative rheumatoid factor.

What is the most likely diagnosis?

Juvenile idiopathic arthritis	8%
Seronegative rheumatoid arthritis	12%
Psoriatic arthritis	75%
Septic arthritis	3%
Rheumatoid arthritis	2%

Psoriatic arthropathy can present before psoriatic skin lesions - a positive family history of psoriasis may point towards this diagnosis

Important for me Less important

Given the inflammatory pattern of pain and swelling (worse in the morning and after rest), the joints involved (DIPs and knee) and family history of psoriasis, the most likely diagnosis is psoriatic arthritis. Psoriatic arthropathy can present before psoriatic skin changes. Therefore, option 3 is the correct answer.

Rheumatoid arthritis and seronegative rheumatoid arthritis classically affect the metacarpal phalangeal joints (MCPs) of the hands, not the DIPs. You would also expect the rheumatoid factor to be raised in rheumatoid arthritis (but not seronegative rheumatoid arthritis).

Idiopathic juvenile arthritis presents like rheumatoid arthritis in patients under 16 years old. Given the age of this patient, this is not a likely diagnosis.

Septic arthritis presents with a swollen, red and very painful joint. The patient would classically be pyrexial and unwell. Therefore, this is not the most likely diagnosis.

Therefore, option 3 is the most likely diagnosis.



Next question >

# Psoriatic arthropathy \*

Psoriatic arthropathy is an inflammatory arthritis associated with psoriasis and is classed as one of the seronegative spondyloarthropathies. It correlates poorly with cutaneous psoriasis and often precedes the development of skin lesions. Around 10-20% of patients with skin lesions develop an arthropathy with males and females being equally affected.

### **Presentation**

#### **Patterns**

- symmetric polyarthritis
  - very similar to rheumatoid arthritis
  - o 30-40% of cases, most common type
- asymmetrical oligoarthritis: typically affects hands and feet (20-30%)
  - until recently it was thought asymmetrical oligoarthritis was the most common type,
     based on data from the original 1973 Moll and Wright paper. Please see the link for a comparison of more recent studies
- sacroiliitis
- DIP joint disease (10%)
- arthritis mutilans (severe deformity fingers/hand, 'telescoping fingers')

# Other signs

- psoriatic skin lesions
- periarticular disease tenosynovitis and soft tissue inflammation resulting in:
  - enthesitis: inflammation at the site of tendon and ligament insertion e.g. Achilles tendonitis, plantar fascitis
  - o tenosynovitis: typically of the flexor tendons of the hands

- o dactylitis: diffuse swelling of a finger or toe
- nail changes
  - pitting
  - o onycholysis

# **Investigation and management**

## X-ray

- often have the unusual combination of coexistence of erosive changes and new bone formation
- periostitis
- 'pencil-in-cup' appearance

# Management

- should be managed by a rheumatologist
- treatment is similar to that of rheumatoid arthritis (RA). However, the following differences are noted:
  - mild peripheral arthritis/mild axial disease may be treated with 'just' an NSAID, rather than all patients being on disease-modifying therapy as with RA
  - o if more moderate/severe disease then methotrexate is typically used as in RA
  - use of monoclonal antibodies such as ustekinumab (targets both IL-12 and IL-23) and secukinumab (targets IL-17)
  - o apremilast: phosphodiesterase type-4 (PDE4) inhibitor → suppression of proinflammatory mediator synthesis and promotion of anti-inflammatory mediators
  - o has a better prognosis than RA









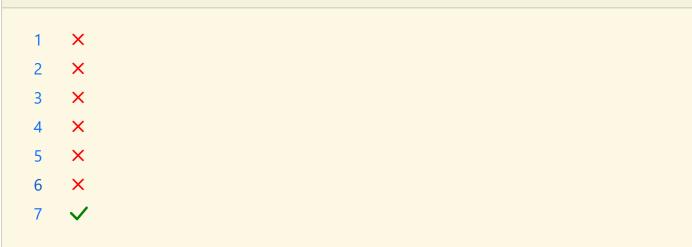


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# High-yield textbook Extended textbook Links 占 11 🖓 11 Annals of the Rheumatic Diseases Relative incidence of polyarthritis vs. oligoarthritis ტ 9 ♀ 7 Patient.info Psoriatic arthropathy review Suggest link Report broken link Media Psoriatic arthritis Zero To Finals - YouTube 677 7 1 Report broken media Score: 14%

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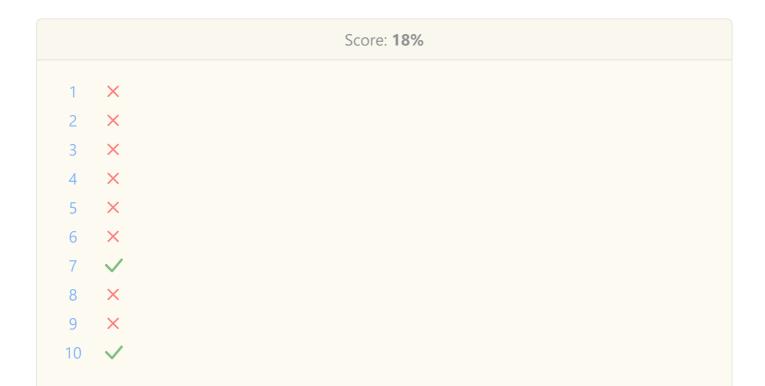
A 65-year-old man presents with lower back and buttock pain which has slowly worsened over the past 6 months He reports a burning pain which is not relieved by paracetamol. He reports the pain has become so severe he often has to sit down when walking downhill to the bus stop. He has no problems walking home from the bus stop and no bladder or bowel dysfunction.

What is the most likely diagnosis?

Prolapsed lumbar disc	
Osteoarthritis	
Sciatica	
Spinal stenosis	
Spinal cord compression	

Submit answer

Reference ranges ∨



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Question 59 of 178



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What is the most likely diagnosis?



Spinal stenosis is often relieved by sitting down or leaning forward

Important for me Less important

Spinal stenosis is the correct answer here as his symptoms are relieved by sitting down and are worse on walking downhill. Spinal stenosis is most common between in the 6th and 7th decades and can present on a background of lower back pain. Leaning forward opens up the spinal canal and can relieve symptoms. Patients are often said to have a characteristic flexed or stooped position when walking. As the condition progresses patients may also report numbness or tingling in the back, buttocks and leg as well as pain.



Next question >

# Lumbar spinal stenosis \*

Lumbar spinal stenosis is a condition in which the central canal is narrowed by tumour, disk prolapse or other similar degenerative changes.

Patients may present with a combination of back pain, neuropathic pain and symptoms mimicking

claudication. One of the main features that may help to differentiate it from true claudication in the history is the positional element to the pain. Sitting is better than standing and patients may find it easier to walk uphill rather than downhill. The neurogenic claudication type history makes lumbar spinal stenosis a likely underlying diagnosis, the absence of such symptoms makes it far less likely.

# **Pathology**

Degenerative disease is the commonest underlying cause. Degeneration is believed to begin in the intervertebral disk where biochemical changes such as cell death and loss of proteoglycan and water content lead to progressive disk bulging and collapse. This process leads to an increased stress transfer to the posterior facet joints, which accelerates cartilaginous degeneration, hypertrophy, and osteophyte formation; this is associated with thickening and distortion of the ligamentum flavum. The combination of the ventral disk bulging, osteophyte formation at the dorsal facet, and ligamentum flavum hyptertrophy combine to circumferentially narrow the spinal canal and the space available for the neural elements. The compression of the nerve roots of the cauda equina leads to the characteristic clinical signs and symptoms of lumbar spinal stenosis.

# **Diagnosis**

MRI scanning is the best modality for demonstrating the canal narrowing. Historically a bicycle test was used as true vascular claudicants could not complete the test.

# **Treatment**

Laminectomy



Next question >





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A 52-year-old woman attends the emergency department following a fall down the bottom 2 steps of her staircase and sustaining an injury to her left arm. She denies any pre-syncopal symptoms but describes feeling generally more lethargic over the last few weeks with generalised body aches and cramps. Her past medical history includes chronic kidney disease stage 1 and she takes no regular medications.

An X-ray of her left forearm confirms a fracture of the distal radius.

# Laboratory tests:

НЬ	124 g/L	(115 - 160)
Platelets	233 * 10 <sup>9</sup> /L	(150 - 400)
WBC	9.6 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	132 mmol/L	(135 - 145)
K <sup>+</sup>	4.1 mmol/L	(3.5 - 5.0)
Urea	7.8 mmol/L	(2.0 - 7.0)
Creatinine	144 µmol/L	(55 - 120)
Calcium	1.8 mmol/L	(2.1-2.6)
Phosphate	0.6 mmol/L	(0.8-1.4)
ALP	126 u/L	(30 - 100)
Parathyroid hormone	14 ng/L	(12-72)
ESR	22 mm/hr	< ((age + 10) / 2)

What is the most likely underlying diagnosis?

Osteomalacia	
Osteopetrosis	
Osteoporosis	
Paget's disease	
Tertiary hyperparathyroidism	

Submit answer

Reference ranges  $\vee$ 

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Question 60 of 178



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A 52-year-old woman attends the emergency department following a fall down the bottom 2 steps of her staircase and sustaining an injury to her left arm. She denies any pre-syncopal symptoms but describes feeling generally more lethargic over the last few weeks with generalised body aches and cramps. Her past medical history includes chronic kidney disease stage 1 and she takes no regular medications.

An X-ray of her left forearm confirms a fracture of the distal radius.

# Laboratory tests:

Hb	124 g/L	(115 - 160)
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WBC	9.6 * 10 <sup>9</sup> /L	(4.0 - 11.0)
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Creatinine	144 µmol/L	(55 - 120)
Calcium	1.8 mmol/L	(2.1-2.6)
Phosphate	0.6 mmol/L	(0.8-1.4)
ALP	126 u/L	(30 - 100)
Parathyroid hormone	14 ng/L	(12-72)
ESR	22 mm/hr	< ((age + 10) / 2)

What is the most likely underlying diagnosis?

Osteomalacia	78%
Osteopetrosis	3%
Osteoporosis	8%
Paget's disease	7%
Tertiary hyperparathyroidism	5%

### Osteomalacia

- low: calcium, phosphate
- raised: alkaline phosphatase

Important for me Less important

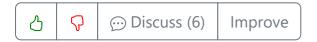
This patient has a diagnosis of **osteomalacia**. Osteomalacia is the demineralisation of bone, most commonly secondary to vitamin D deficiency. Patients with osteomalacia typically present with bony pain, muscle tenderness, proximal myopathy, and an increased tendency for fractures. Blood tests reveal low calcium and phosphate levels and raised alkaline phosphatase levels (ALP), as seen here. This patient describes a history of lethargy and body aches and cramps that is likely a result of her hypocalcaemia.

**Osteopetrosis**, also known as marble bone disease, is a disorder of osteoclast function that impairs bone resorption. This results in dense bones that are likely to fracture. Bone pains are common, like those described by this patient. However, on laboratory testing, calcium, phosphate and ALP levels are normal.

**Osteoporosis** is a reduced bone mineral density associated with multiple risk factors including menopause, drugs (e.g. glucocorticoids), chronic kidney disease and endocrine disorders. Similar to this patient's presentation, osteoporosis also increases the likelihood of fractures. However, unlike this patient, patients with osteoporosis have normal calcium, phosphate and ALP levels.

**Paget's disease** is most commonly seen in men of increasing age. It is usually asymptomatic although can present with bone pain, particularly in the pelvis and lumbar spine. It is uncommon in a young woman such as this patient. Furthermore, laboratory tests typically reveal an isolated raised ALP but with normal calcium and phosphate levels.

**Tertiary hyperparathyroidism** is commonly seen in patients with chronic kidney disease. Initially, there is compensatory hypertrophy of the parathyroid glands secondary to hypocalcaemia. As the disease progresses, persistent secondary hyperparathyroidism causes parathyroid hyperplasia (tertiary hyperparathyroidism). In tertiary hyperparathyroidism, calcium and phosphate are both raised with a raised parathyroid hormone.



Next question >

# Osteomalacia \*

Osteomalacia describes softening of the bones secondary to low vitamin D levels that in turn lead to decreased bone mineral content. If this occurs in growing children it is referred to as rickets, with the term osteomalacia preferred for adults.

### Causes

- vitamin D deficiency
  - o malabsorption
  - lack of sunlight
  - o diet
- chronic kidney disease
- drug induced e.g. anticonvulsants
- inherited: hypophosphatemic rickets (previously called vitamin D-resistant rickets)
- liver disease: e.g. cirrhosis
- coeliac disease

### **Features**

- bone pain
- bone/muscle tenderness
- fractures: especially femoral neck
- proximal myopathy: may lead to a waddling gait

# Investigation

- bloods
  - o low vitamin D levels
  - low calcium, phosphate (in around 30%)
  - o raised alkaline phosphatase (in 95-100% of patients)
- x-ray
  - translucent bands (Looser's zones or pseudofractures)

#### **Treatment**

- vitamin D supplmentation
  - a loading dose is often needed initially
- calcium supplementation if dietary calcium is inadequate



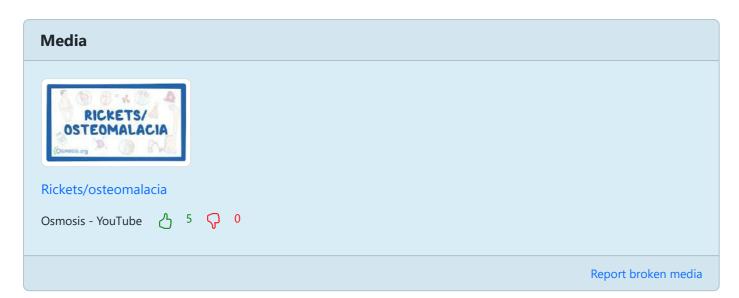
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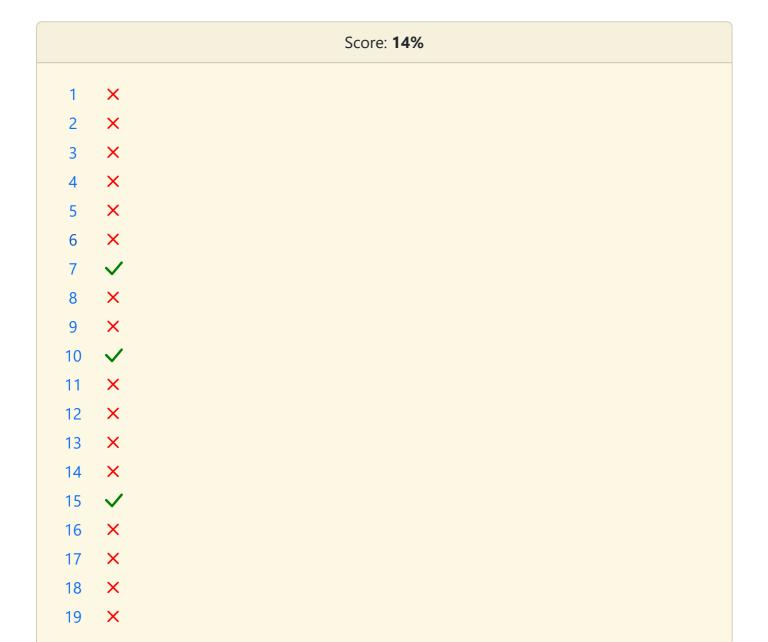


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#### Question 61 of 178



 $\overline{\Rightarrow}$ 

A 48-year-old woman attends with progressive shortness of breath on exertion. On examination, you note coarse bibasal inspiratory crepitations and thickening of the skin of her proximal limbs. Observations are as follows: heart rate 85 beats per minute, respiratory rate 22 breaths per minute, blood pressure 210/95 mmHg, temperature 37.5°C, and oxygen saturation 95% on air. Her urinary output is 0.4 ml/kg/hour.

#### Blood results are as follows:

Hb	105 g/L	Male: (135-180) Female: (115 - 160)
Platelets	154 * 10 <sup>9</sup> /L	(150 - 400)
WBC	15.8 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	132 mmol/L	(135 - 145)
K <sup>+</sup>	4.9 mmol/L	(3.5 - 5.0)
Urea	18.2 mmol/L	(2.0 - 7.0)
Creatinine	426 µmol/L	(55 - 120)
CRP	3 mg/L	(< 5)

ANA	1:128	(<1:40)
Anti dsDNA	68 IU/mL	(<10)

## A chest x-ray is performed:

Chest x-ray Bilateral air space opacification with diversion of blood to the upper lobes

#### What treatment is indicated?

Dialysis	
Losartan	
Prostacyclin	
Ramipril	
Steroids and mycophenolate	

# Submit answer

Reference ranges  $\checkmark$ 

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Question 61 of 178



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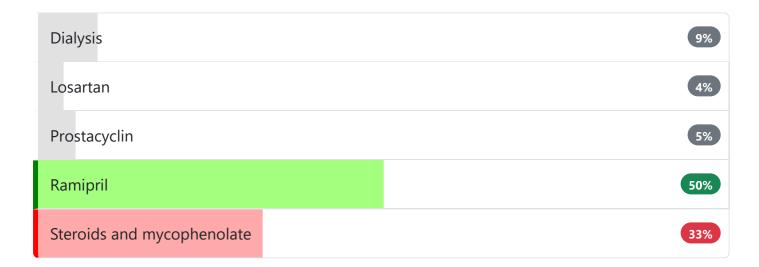
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CRP	3 mg/L	(< 5)

ANA	1:128	(<1:40)
Anti dsDNA	68 IU/mL	(<10)

### A chest x-ray is performed:

Chest x-ray Bilateral air space opacification with diversion of blood to the upper lobes

#### What treatment is indicated?





The clinical presentation is of diffuse cutaneous systemic sclerosis with a scleroderma renal crisis (SRC). SRC is characterised by malignant hypertension and oligo-anuric acute renal failure. A renal biopsy is not necessary to confirm the diagnosis of classical SRC.

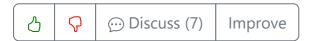
**Ramipril** is correct. ACE inhibitors (ACEi) are the mainstay of therapy in SRC. Initiation of their use allowed for major increased survival rates of SRC over the last decades. The treatment goal is to achieve blood pressure control as soon as possible. Importantly, ACE inhibitors must be continued even if renal function is deteriorating.

**Dialysis** is incorrect. Indications for dialysis include uraemic encephalopathy, uraemic pericarditis, uraemic haemorrhage, oliguria resulting in clinically significant volume overload and respiratory distress (that is refractory to medical management), metabolic acidosis due to renal failure, and hyperkalaemia (>6.0) refractory to medical management. The patient does not fulfil any of these criteria at the current time.

**Losartan** is incorrect. Angiotensin II receptor blockade (ATII) may be useful but is anecdotally less effective than ACE inhibitors, although clinical experience with these agents has been limited.

**Prostacyclin** is incorrect. Continuous low doses of prostacyclin may be added to standard therapy although there is no evidence that it improves short-term and long-term prognosis.

**Steroids and mycophenolate** is incorrect. Steroids have been shown to precipitate and worsen SRC. Clinical trials have yet to demonstrate the efficacy of immunosuppressive agents for the treatment of SRC.



Next question >

# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

Raynaud's may be the first sign

- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

### Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - patients with renal disease should be started on an ACE inhibitor
- poor prognosis

### Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







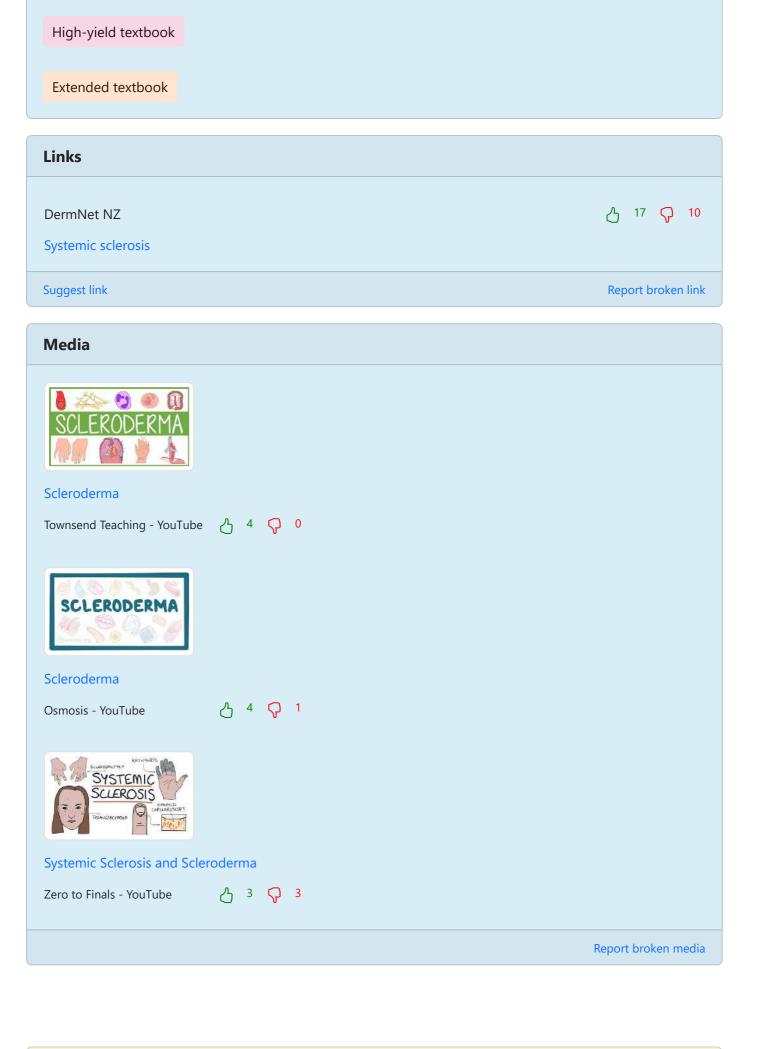
#### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis



Next question >





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#### Question 62 of 178

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 $\Rightarrow$ 

A 74-year-old woman attends rheumatology clinic for a review of her osteoporosis treatment. The patient had been diagnosed with osteoporosis on a DEXA scan five years previously after she fell and sustained a Colles fracture on the left side. Following this diagnosis, the patient had been initiated on treatment with alendronic acid.

In clinic, the patient reported that she had recently been suffering from nagging back pain over the past few weeks. She denied any history of recent falls or other trauma.

Past medical history was significant for rheumatoid arthritis, diagnosed when the patient was 28 years old. Following this diagnosis, she had received prolonged treatment with corticosteroids in association with a variety of disease modifying drugs. Ultimately, good control of her arthritis had been achieved using methotrexate (10 mg weekly) and the patient had not required corticosteroid treatment for many years. The patient reported no family history of osteoporosis or fragility fractures. She did not smoke or drink any alcohol.

The patient reported no concerns or side effects associated with taking her weekly alendronic acid (70 mg weekly).

Examination of the patients spine demonstrated mid-line point tenderness around the T12 - L1 level. Neurological examination of the lower limbs was unremarkable.

Thoracolumbar spine x-ray: anterior height loss of T12 vertebrae, otherwise unremarkable

Height	150 cm
Weight	55 kg
Femoral neck BMD (5 years previously)	T - 3.2
Femoral neck BMD (present day)	T - 2.4
FRAX 10-year probability of major osteoporotic fracture	27 %
FRAX 10-year probability of hip fracture	8.7 %

What is the most appropriate management of the patient's osteoporosis?

Hold further osteoporosis treatment with repeat DEXA scan in two years	
Hold further osteoporosis treatment with repeat DEXA scan in five years	
Discontinue alendronic acid and initiate treatment with denosumab	

Discontinue alendronic acid and initiate treatment with zoledronic acid

 Continue treatment with alendronic acid with repeat DEXA scan in five years

# Submit answer

Reference ranges ✓

## Score: **18%** X 2 X 3 X 4 X 5 X X 6 7 8 X 9 X 10 11 X X 12 X 13 X 14 15 16 X X 17 X 18 19 X 20 X 21 X 22 X 24 25

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Ouestion 62 of 178



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A 74-year-old woman attends rheumatology clinic for a review of her osteoporosis treatment. The patient had been diagnosed with osteoporosis on a DEXA scan five years previously after she fell and sustained a Colles fracture on the left side. Following this diagnosis, the patient had been initiated on treatment with alendronic acid.

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Femoral neck BMD (present day)	T - 2.4
FRAX 10-year probability of major osteoporotic fracture	27 %
FRAX 10-year probability of hip fracture	8.7 %

What is the most appropriate management of the patient's osteoporosis?

Hold further osteoporosis treatment with repeat DEXA scan in two years

Hold further osteoporosis treatment with repeat DEXA scan in five years

Discontinue alendronic acid and initiate treatment with denosumab

34%



Continue treatment with alendronic acid with repeat DEXA scan in five years

34%

The patient's bone mineral density has improved secondary to her treatment with alendronic acid. However, her recent back pain and abnormal thoracolumbar x-ray suggest she has suffered from a osteoporotic vertebral fracture. In one study, continuing alendronic acid from five to ten years treatment reduced the incidence of clinical vertebral fractures in all patients regardless of T score. Therefore, continuing alendronic acid treatment would be recommended for this patient.

Due to increased awareness of the potential complications of long-term bisphosphonate treatment, treatment breaks (or 'drug holidays') are now employed for some patients. Specifically, a treatment break should be considered for patients less than 75 years old, with femoral neck T score > - 2.5, no history of osteoporotic vertebral fracture and deemed low risk following assessment by WHO Fracture Risk Assessment Tool (FRAX) and National Osteoporosis Guideline Group (NOGG) guidance. For such a patient, treatment would typically be suspended for two years with repeat DEXA scan at the end of that period, or sooner in the event of fragility fracture.

Parenteral osteroporosis treatments such as zoledronic acid or denosumab should be considered if patient is intolerant of oral bisphosphonates or in the event of treatment failure (defined as two or more fractures on treatment, or one fracture and a fall in bone mineral density).

Paskins Z, Warburton L. Bisphosphonates beyond five years. BMJ 2016;352:i264.

https://www.shef.ac.uk/FRAX/tool.jsp



Next question >

# Osteoporosis: management \*

The National Osteoporosis Guideline Group (NOGG) updated their guidelines in 2021. NICE guidelines also have a section on the management of osteoporosis, largely based on the NOGG guidelines. Remember that osteoporosis is usually asymptomatic until a fracture occurs. When thinking about osteoporosis management it is useful to think about a number of potential clinical scenarios:

- a patient who has been identified as being at high risk of a fragility fracture based on a QFracture or FRAX score (please see the textbook entry on 'Osteoporosis: assessing risk')
- a patient who is about to start treatment that puts them at significant risk of developing osteoporosis the most common example is longer-term glucocorticoids
- a patient who has just had a fragility fracture e.g. a symptomatic osteoporotic vertebral fracture

# **General management points**

General points about the management of all patients

- all patients who are at risk of osteoporosis or have osteoporosis should be given advice regarding:
  - lifestyle changes: a healthy, balanced diet, moderation of alcohol consumption and avoidance of smoking
  - o a sufficient dietary calcium and vitamin D intake: supplementation should be offered to all women unless the clinician is confident they have adequate calcium intake and are vitamin D replete
  - o encourage a combination of regular weight-bearing and muscle strengthening exercise
- secondary causes of osteoporosis should be considered and treated
  - e.g. hypogonadism in women or men e.g. hormone replacement therapy for premature menopause
- bisphosphonates are the first-line drug treatment for patients at risk of fragility fractures
  - oral bisphosphonates such as alendronate and risedronate are typically first-line. These are often taken weekly are need taking in a particular way to minimise the risk of oesophageal side-effects
  - however, the NOGG recommend IV zoledronate as the first-line treatment following a hip fracture. This is given yearly
- denosumab is generally used as a second-line treatment
- other possible treatment options include:
  - o strontium ranelate
  - raloxifene
  - o teriparatide
  - o romosozumab

### Clinical scenarios

Fragility risk fracture assessment

- if a patient is deemed high-risk based on a QFracture or FRAX score they should have a DEXA scan to assess bone mineral density (BMD)
  - the BMD threshold for defining osteoporosis is a T-score of 2.5 SD or below
  - o some patients may not be suitable for BMD assessment due to frailty etc.
- general osteoporosis management as above
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

Postmenopausal women, and men age ≥50, who are treated with oral glucocorticoids:

- if starting ≥7.5 mg/day prednisolone or equivalent for the next 3 months, start bone protective treatment at the same time
- general osteoporosis management as above
- don't wait for a DEXA scan before starting treatment
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

A postmenopausal woman, or a man age ≥50 has a symptomatic osteoporotic vertebral fracture:

- general osteoporosis management as above
- start treatment straight away oral bisphosphonates are used first-line e.g. alendronate or risedronate

#### Hip fracture in older adults

- in older adults a hip fracture is a manifestation of osteoporosis
  - o following a fragility fracture in women ≥ 75 years, a DEXA scan is not necessary to diagnose osteoporosis and hence commence a bisphosphonate
  - BMD should be measured, but this acts as a baseline rather than determining whether treatment should be given
- bisphosphonates should be given first-line
  - NOGG recommends IV zoledronate but local guidelines may vary and oral bisphosphonates are often used

## Follow-up

Plan to prescribe oral bisphosphonates for at least 5 years, or intravenous bisphosphonates for at least 3 years and then re-assess fracture risk.

# Supplementary notes on treatment

### **Bisphosphonates**

- bisphosphonates bind to hydroxyapatite in bone, inhibiting osteoclast-mediated bone resorption
- common side effects include gastrointestinal discomfort, oesophagitis, and hypocalcaemia. Atypical femoral fractures and osteonecrosis of the jaw are rare but serious risks.
- available in oral and intravenous formulations. Oral bisphosphonates should be taken with a full glass of water, on an empty stomach, and the patient should remain upright for at least 30 minutes afterwards.

#### Denosumab

- human monoclonal antibody that inhibits RANK ligand, which in turn inhibits the maturation of osteoclasts
- also used for cancer patients with bone metastases to reduce skeletal-related events.
- given as a single subcutaneous injection every 6 months

#### Raloxifene

- selective oestrogen receptor modulator (SERM)
- has been shown to prevent bone loss and to reduce the risk of vertebral fractures, but has not yet been shown to reduce the risk of non-vertebral fractures
- has been shown to increase bone density in the spine and proximal femur

- may worsen menopausal symptoms
- increased risk of thromboembolic events
- may decrease the risk of breast cancer

#### Strontium ranelate

- 'dual action bone agent' increases deposition of new bone by osteoblasts (promotes differentiation of pre-osteoblast to osteoblast) and reduces the resorption of bone by inhibiting osteoclasts
- concerns regarding the safety profile of strontium have been raised recently. It should only be prescribed by a specialist in secondary care
- due to these concerns the European Medicines Agency in 2014 said it should only be used by people for whom there are no other treatments for osteoporosis
- increased risk of cardiovascular events: any history of cardiovascular disease or significant risk of cardiovascular disease is a contraindication
- increased risk of thromboembolic events: a Drug Safety Update in 2012 recommended it is not used in patients with a history of venous thromboembolism
- may cause serious skin reactions such as Stevens Johnson syndrome

### **Teriparatide**

- recombinant form of parathyroid hormone
- very effective at increasing bone mineral density but role in the management of osteoporosis yet to be clearly defined

#### Romosozumab

- a monoclonal antibody that inhibits sclerostin, thereby increasing bone formation and decreasing bone resorption
- this dual action significantly improves bone density and reduces fracture risk.



© Image used on license from Radiopaedia

MRI showing osteoporotic fractures of the 10th and 12th thoracic vertebrae.



Next question >

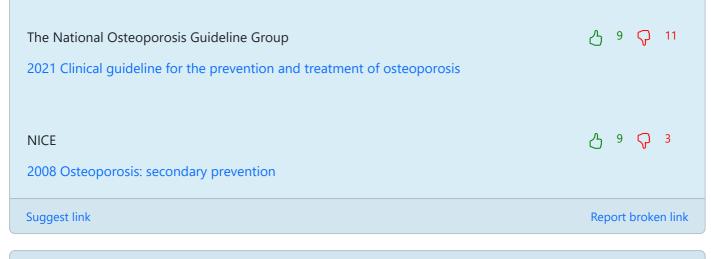


## **Textbooks**

High-yield textbook

Extended textbook

## Links



## Media



Osteoporosis pharmacology, prevention and treatment

Armando Hasudungan - YouTube







Report broken media

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#### Question 63 of 178

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A 73-year-old woman attends rheumatology clinic for review of her osteoporosis treatment. The patient had been diagnosed with osteoporosis five years previously on a DEXA scan performed after she had sustained a fractured right neck of femur. Since the time of diagnosis, the patient had been receiving treatment with alendronic acid (70 mg weekly).

During clinic review, the patient reported taking her alendronic acid as prescribed without any associated side effects. A review of her past medical history revealed that the patient had sustained a left distal radius fracture the previous year following a trip at home. In addition, the patient had suffered from a deep vein thrombosis in her right leg three years before precipitated by a trans-continental flight and had been anti-coagulated with warfarin for six months.

The patient was a smoker (10 cigarettes per day) and also consumed around 20 units of alcohol per week. Her mother had suffered a fractured neck of femur at the age of 80. The patient had never been diagnosed with rheumatoid arthritis and had no significant exposure to corticosteroid treatment.

Clinical examination of the patient demonstrated no loss of height since previous measurement five years previously. There was no tenderness on palpation of the thoracic or lumbar spine.

Height	175 cm
Weight	95 kg
Femoral neck BMD (5 years previously)	T - 2.7
Femoral neck BMD (present day)	T - 2.9
FRAX 10-year probability of major osteoporotic fracture	60 %
FRAX 10-year probability of hip fracture	50 %

What is the appropriate management of the patient's osteoporosis?

Stop treatment with alendronic acid and start treatment with denosumab	
Stop treatment with alendronic acid and start treatment with strontium ranelate	
Continue treatment with alendronic acid with repeat DEXA scan in five years	
Continue treatment with alendronic acid with repeat DEXA scan in two years	
Stop treatment with alendronic acid with repeat DEXA scan in two years	

# Submit answer

Reference ranges  $\checkmark$ 

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### Score: **18%** X 1 2 X 3 X 4 × 5 X 6 X 7 X 8 9 X 10 X 11 X 12 X 13 14 X 15 **/** 16 × 17 X 18 X 19 × 20 21 X 22 X 23 X × 24 25 26 × 27 X 28



Question 63 of 178



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A 73-year-old woman attends rheumatology clinic for review of her osteoporosis treatment. The patient had been diagnosed with osteoporosis five years previously on a DEXA scan performed after she had sustained a fractured right neck of femur. Since the time of diagnosis, the patient had been receiving treatment with alendronic acid (70 mg weekly).

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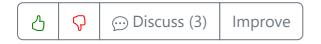
Stop treatment with alendronic acid and start treatment with denosumab	52%
Stop treatment with alendronic acid and start treatment with strontium ranelate	6%
Continue treatment with alendronic acid with repeat DEXA scan in five years	13%
Continue treatment with alendronic acid with repeat DEXA scan in two years	23%
Stop treatment with alendronic acid with repeat DEXA scan in two years	5%

Due to her extensive risk factors, the patient is at an extremely high risk of suffering from further fragility fractures and it is essential that she continues with osteoporosis treatment. Bisphosphonate treatment failure is defined as two or more fractures on treatment, or one fracture with a reduction in bone density (as in this patient). Therefore, continuing treatment with alendronic acid is not advisable. Of the alternative treatments listed as possible answers, strontium ranelate is contra-indicated in this patient due to her history of deep vein thrombosis, leading to treatment with denosumab as the most appropriate treatment option.

Due to increasing awareness of complications associated with long-term bisphosphonate treatment, treatment breaks (or 'drug holidays') can be considered for some patient's after five years treatment. Guidelines recommend consideration of treatment breaks for patients less than 75 years old with a femoral neck T-score greater than -2.5 and who are defined as low risk by WHO Fracture Risk Assessment Tool (FRAX) and National Osteoporosis Guideline Group (NOGG) guidelines. When a drug holiday is agreed, advice is to repeat DEXA scan after two years, or sooner in the event of a fracture. Treatment breaks are never recommended if a patient has ever suffered from a vertebral insufficiency fracture.

Paskins Z, Warburton L. Bisphosphonates beyond five years. BMJ 2016;352:i264.

https://www.shef.ac.uk/FRAX/tool.jsp



Next question >

# Osteoporosis: management \*

The National Osteoporosis Guideline Group (NOGG) updated their guidelines in 2021. NICE guidelines also have a section on the management of osteoporosis, largely based on the NOGG guidelines. Remember that osteoporosis is usually asymptomatic until a fracture occurs. When thinking about osteoporosis management it is useful to think about a number of potential clinical scenarios:

- a patient who has been identified as being at high risk of a fragility fracture based on a QFracture or FRAX score (please see the textbook entry on 'Osteoporosis: assessing risk')
- a patient who is about to start treatment that puts them at significant risk of developing osteoporosis the most common example is longer-term glucocorticoids
- a patient who has just had a fragility fracture e.g. a symptomatic osteoporotic vertebral fracture

# **General management points**

General points about the management of all patients

- all patients who are at risk of osteoporosis or have osteoporosis should be given advice regarding:
  - lifestyle changes: a healthy, balanced diet, moderation of alcohol consumption and avoidance of smoking
  - o a sufficient dietary calcium and vitamin D intake: supplementation should be offered to all women unless the clinician is confident they have adequate calcium intake and are vitamin D replete
  - o encourage a combination of regular weight-bearing and muscle strengthening exercise
- secondary causes of osteoporosis should be considered and treated
  - e.g. hypogonadism in women or men e.g. hormone replacement therapy for premature menopause
- bisphosphonates are the first-line drug treatment for patients at risk of fragility fractures
  - oral bisphosphonates such as alendronate and risedronate are typically first-line. These are often taken weekly are need taking in a particular way to minimise the risk of oesophageal side-effects
  - however, the NOGG recommend IV zoledronate as the first-line treatment following a hip fracture. This is given yearly
- denosumab is generally used as a second-line treatment
- other possible treatment options include:
  - o strontium ranelate
  - raloxifene
  - o teriparatide
  - romosozumab

### Clinical scenarios

Fragility risk fracture assessment

- if a patient is deemed high-risk based on a QFracture or FRAX score they should have a DEXA scan to assess bone mineral density (BMD)
  - the BMD threshold for defining osteoporosis is a T-score of 2.5 SD or below
  - o some patients may not be suitable for BMD assessment due to frailty etc.
- general osteoporosis management as above
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

Postmenopausal women, and men age ≥50, who are treated with oral glucocorticoids:

- if starting ≥7.5 mg/day prednisolone or equivalent for the next 3 months, start bone protective treatment at the same time
- general osteoporosis management as above
- don't wait for a DEXA scan before starting treatment
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

A postmenopausal woman, or a man age  $\geq$ 50 has a symptomatic osteoporotic vertebral fracture:

• general osteoporosis management as above

 start treatment straight away - oral bisphosphonates are used first-line e.g. alendronate or risedronate

#### Hip fracture in older adults

- in older adults a hip fracture is a manifestation of osteoporosis
  - following a fragility fracture in women ≥ 75 years, a DEXA scan is not necessary to diagnose osteoporosis and hence commence a bisphosphonate
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- bisphosphonates should be given first-line
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## Follow-up

Plan to prescribe oral bisphosphonates for at least 5 years, or intravenous bisphosphonates for at least 3 years and then re-assess fracture risk.

# Supplementary notes on treatment

### Bisphosphonates

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- given as a single subcutaneous injection every 6 months

#### Raloxifene

- selective oestrogen receptor modulator (SERM)
- has been shown to prevent bone loss and to reduce the risk of vertebral fractures, but has not yet been shown to reduce the risk of non-vertebral fractures
- has been shown to increase bone density in the spine and proximal femur
- may worsen menopausal symptoms
- increased risk of thromboembolic events
- may decrease the risk of breast cancer

#### Strontium ranelate

- 'dual action bone agent' increases deposition of new bone by osteoblasts (promotes differentiation of pre-osteoblast to osteoblast) and reduces the resorption of bone by inhibiting osteoclasts
- concerns regarding the safety profile of strontium have been raised recently. It should only be prescribed by a specialist in secondary care
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- increased risk of thromboembolic events: a Drug Safety Update in 2012 recommended it is not used in patients with a history of venous thromboembolism
- may cause serious skin reactions such as Stevens Johnson syndrome

### Teriparatide

- recombinant form of parathyroid hormone
- very effective at increasing bone mineral density but role in the management of osteoporosis yet to be clearly defined

#### Romosozumab

- a monoclonal antibody that inhibits sclerostin, thereby increasing bone formation and decreasing bone resorption
- this dual action significantly improves bone density and reduces fracture risk.



© Image used on license from Radiopaedia

MRI showing osteoporotic fractures of the 10th and 12th thoracic vertebrae.



Next question >

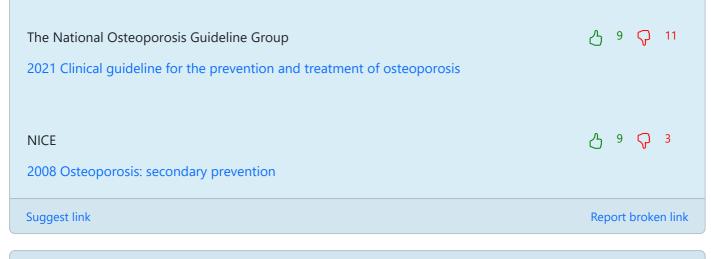


## **Textbooks**

High-yield textbook

Extended textbook

## Links



## Media



Osteoporosis pharmacology, prevention and treatment

Armando Hasudungan - YouTube







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A 52-year-old man presents with lethargy and reduced sensation in both feet. He reports a 2 month history of fevers and 4kg weight loss. He also reports intermittent testicular pain.

On examination there is livedo reticularis on both legs and reduced light touch and pain sensation on both feet.

#### Blood tests reveal:

Hb	116 g/l	Na <sup>137</sup>	# mmol/l	Bilirubin	18 µmol/l
Platelets	487 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.8 mmol/l	ALP	92 u/l
WBC	8.3 * 10 <sup>9</sup> /l	Urea	12.8 mmol/l	ALT	102 u/l
Neuts	6.3 * 10 <sup>9</sup> /l	Creatinine	182 µmol/l	γGT	16 u/l
MCV	89 fL	ESR	78mm/hr	Albumin	34 g/l

Which investigation is most likely to reveal the diagnosis?

Hepatitis C serology	
Renal angiogram	
cANCA	
pANCA	
Hepatitis B serology	

Submit answer

Reference ranges ∨

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Question 64 of 178



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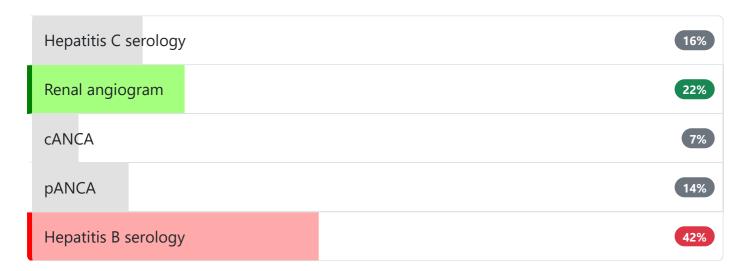
A 52-year-old man presents with lethargy and reduced sensation in both feet. He reports a 2 month history of fevers and 4kg weight loss. He also reports intermittent testicular pain.

On examination there is livedo reticularis on both legs and reduced light touch and pain sensation on both feet.

#### Blood tests reveal:

Hb	116 g/l	Na <sup>137</sup>	# mmol/l	Bilirubin	18 µmol/l
Platelets	487 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.8 mmol/l	ALP	92 u/l
WBC	8.3 * 10 <sup>9</sup> /l	Urea	12.8 mmol/l	ALT	102 u/l
Neuts	6.3 * 10 <sup>9</sup> /l	Creatinine	182 µmol/l	γGT	16 u/l
MCV	89 fL	ESR	78mm/hr	Albumin	34 g/l

Which investigation is most likely to reveal the diagnosis?



Fever, lethargy, neuropathy, testicular pain and renal dysfunction are consistent with polyarteritis nodosa (PAN).

Whilst the other investigations listed would be appropriate, angiography would be most likely to confirm the diagnosis (sensitivity 89%, specificity 90%).

ANCA is classically negative in PAN. Hepatitis serology would be appropriate as this can be associated with vasculitis however will not reveal the diagnosis in this case.

(BMJ BestPractice)

# Polyarteritis nodosa 🖈

Polyarteritis nodosa (PAN) is a vasculitis affecting medium-sized arteries with necrotizing inflammation leading to aneurysm formation. PAN is more common in middle-aged men and is associated with hepatitis B infection.

#### **Features**

- fever, malaise, arthralgia
- weight loss
- hypertension
- mononeuritis multiplex, sensorimotor polyneuropathy

Improve

- testicular pain
- livedo reticularis
- haematuria, renal failure
- perinuclear-antineutrophil cytoplasmic antibodies (ANCA) are found in around 20% of patients with 'classic' PAN
- hepatitis B serology positive in 30% of patients



© Image used on license from DermNet NZ



© Image used on license from Radiopaedia

Angiogram from a patient with polyarteritis nodosa. Both kidneys demonstrate beading and numerous microaneurysms affecting the intrarenal vessels. Similar changes are seen affecting the intrahepatic vessels with a few small microaneurysms noted. The proximal branches of the SMA appears normal; however there are no normal straight arteries from the jejunal arteries and lack of normal anastomotic arcades and loops. This is associated with multiple microaneurysms.

Next question >



# **Textbooks**

High-yield textbook

Extended textbook

#### Media



Polyarteritis Nodosa and Kawasaki Disease (Medium Vessel Vasculitis) - Symptoms, pathophysiology

Armando Hasudungan - YouTube

Report broken media

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A 40-year-old woman is seen in an outpatient rheumatology clinic. She was diagnosed with Systemic lupus erythematosus 5 years previously when she has presented with fatigue, anaemia and a rash. She also has a past medical history of hypertension, gout and psoriasis.

She had noticed that the joints in her hands were becoming deformed, however, she was able to complete day to day task without any functional impairment and denied pain in the affected joints.

On examination, she had symmetrical marked reducible ulnar subluxation and deviation at the MCP joints. X-rays of her hands showed no erosions.

What is the most likely diagnosis?

Jaccoud's arthropathy	
Rheumatoid arthritis	
Gout	
Psoriatic arthritis	
Sarcoid arthropathy	

Submit answer

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Question 65 of 178



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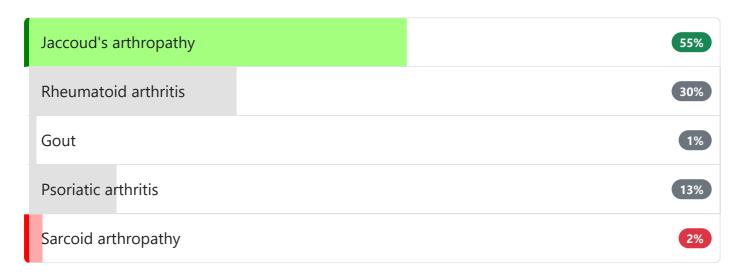


A 40-year-old woman is seen in an outpatient rheumatology clinic. She was diagnosed with Systemic lupus erythematosus 5 years previously when she has presented with fatigue, anaemia and a rash. She also has a past medical history of hypertension, gout and psoriasis.

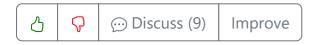
She had noticed that the joints in her hands were becoming deformed, however, she was able to complete day to day task without any functional impairment and denied pain in the affected joints.

On examination, she had symmetrical marked reducible ulnar subluxation and deviation at the MCP joints. X-rays of her hands showed no erosions.

What is the most likely diagnosis?



The key here is the absence of pain which is more than likely to be present in options B to E hence the presence of psoriasis is a distracting factor. Additionally, gout usually affects the first metatarsal head and sarcoid arthropathy is very rare hence Jaccoud's arthropathy which is none erosive and associated with systemic lupus erythematosus is the correct answer.



Next question >

# Systemic lupus erythematosus: features \*

Systemic lupus erythematosus (SLE) is a multisystem, autoimmune disorder. It typically presents in early adulthood and is more common in women and people of Afro-Caribbean origin.

#### General features

fatigue

- fever
- mouth ulcers
- lymphadenopathy

#### Skin

- malar (butterfly) rash: spares nasolabial folds
- discoid rash: scaly, erythematous, well demarcated rash in sun-exposed areas. Lesions may progress to become pigmented and hyperkeratotic before becoming atrophic
- photosensitivity
- Raynaud's phenomenon
- livedo reticularis
- non-scarring alopecia

#### Musculoskeletal

- arthralgia
- non-erosive arthritis

#### Cardiovascular

- pericarditis: the most common cardiac manifestation
- myocarditis

## Respiratory

- pleurisy
- fibrosing alveolitis

#### Renal

- proteinuria
- glomerulonephritis (diffuse proliferative glomerulonephritis is the most common type)

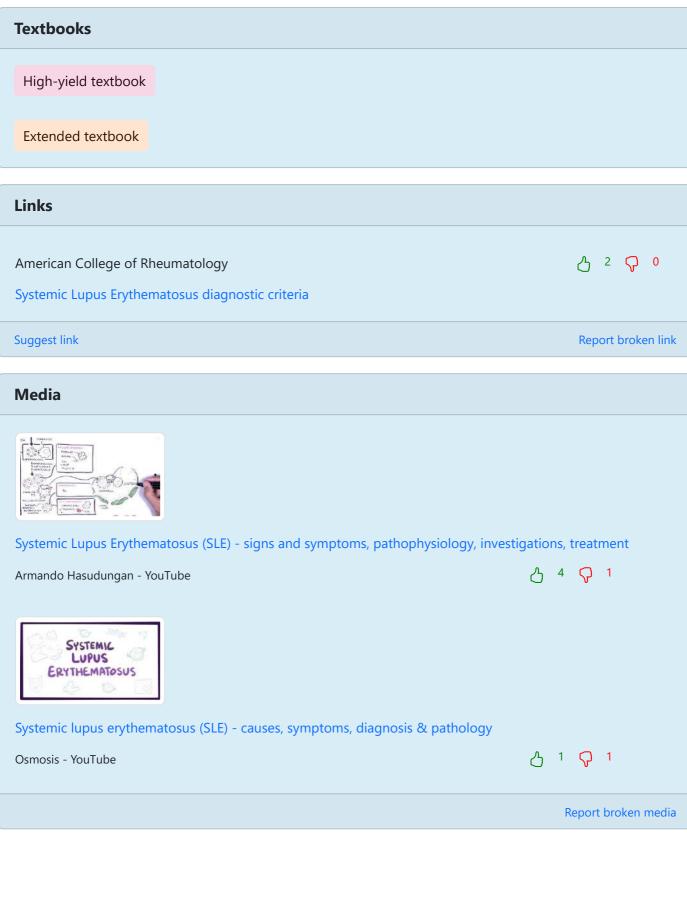
### Neuropsychiatric

- anxiety and depression
- psychosis
- seizures



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Question 66 of 178





This patient is known to suffer from Raynaud's phenomenon:



What does the lesion on her thumb most likely represent?

Arterial ulcer	
Gouty tophus	×
Calcium deposit	×
Orf	×
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Submit answer

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Question 66 of 178



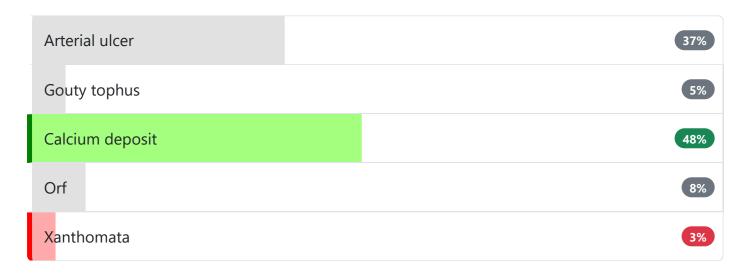
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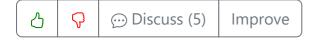
This patient is known to suffer from Raynaud's phenomenon:



What does the lesion on her thumb most likely represent?



This lesion represents calcinosis.



Next question >

# Systemic sclerosis ★

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - o patients with renal disease should be started on an ACE inhibitor
- poor prognosis

Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







#### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis



Next question >



# **Textbooks** High-yield textbook Extended textbook Links DermNet NZ Systemic sclerosis Suggest link Report broken link Media Scleroderma Townsend Teaching - YouTube 4 Q 0 SCLERODERMA Scleroderma Osmosis - YouTube 💍 4 🥎 1 Systemic Sclerosis and Scleroderma Zero to Finals - YouTube

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Question 67 of 178



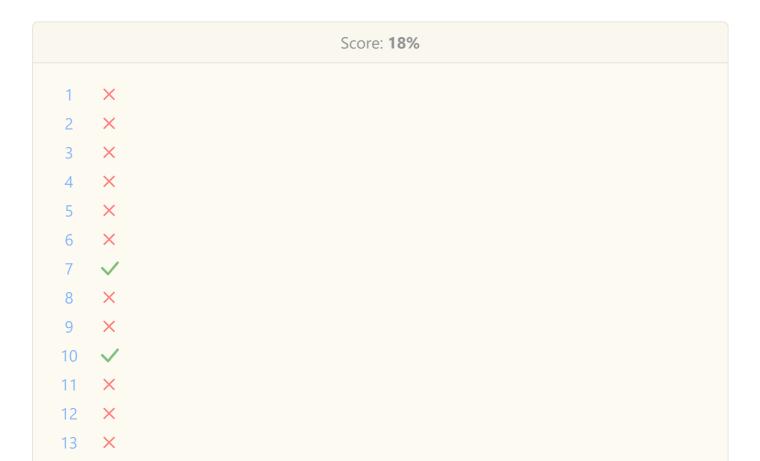


A 75-year-old female was recently started on alendronate for treatment of osteoporosis following a fragility fracture. She returns to your clinic as she has suffered troubling upper gastrointestinal side effects. What is the most appropriate next step in her management?

Continue alendronate	
Change alendronate to strontium ranelate	
Change alendronate to risedronate	
Change alendronate to raloxifene	
Change alendronate to denosumab	

Submit answer

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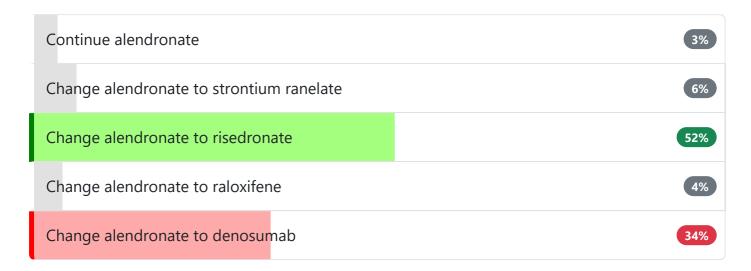




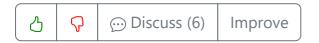




A 75-year-old female was recently started on alendronate for treatment of osteoporosis following a fragility fracture. She returns to your clinic as she has suffered troubling upper gastrointestinal side effects. What is the most appropriate next step in her management?



NICE guidance recommends that if patients suffer significant upper gastrointestinal side effects from the use of alendronate, then this should first be changed to risedronate or etidronate



Next question >

# Osteoporosis: management \*

The National Osteoporosis Guideline Group (NOGG) updated their guidelines in 2021. NICE guidelines also have a section on the management of osteoporosis, largely based on the NOGG guidelines. Remember that osteporosis is usually asymptomatic until a fracture occurs. When thinking about osteoporosis management it is useful to think about a number of potential clinical scenarios:

- a patient who has been identified as being at high risk of a fragility fracture based on a QFracture or FRAX score (please see the textbook entry on 'Osteoporosis: assessing risk')
- a patient who is about to start treatment that puts them at significant risk of developing osteoporosis - the most common example is longer-term glucocorticoids
- a patient who has just had a fragility fracture e.g. a symptomatic osteoporotic vertebral fracture

# **General management points**

General points about the management of all patients

- all patients who are at risk of osteoporosis or have osteoporosis should be given advice regarding:
  - o lifestyle changes: a healthy, balanced diet, moderation of alcohol consumption and avoidance of smoking
  - a sufficient dietary calcium and vitamin D intake: supplementation should be offered to all women unless the clinician is confident they have adequate calcium intake and are vitamin D replete
  - o encourage a combination of regular weight-bearing and muscle strengthening exercise
- secondary causes of osteoporosis should be considered and treated
  - e.g. hypogonadism in women or men e.g. hormone replacement therapy for premature menopause
- bisphosphonates are the first-line drug treatment for patients at risk of fragility fractures
  - oral bisphosphonates such as alendronate and risedronate are typically first-line. These are often taken weekly are need taking in a particular way to minimise the risk of oesophageal side-effects
  - however, the NOGG recommend IV zoledronate as the first-line treatment following a hip fracture. This is given yearly
- denosumab is generally used as a second-line treatment
- other possible treatment options include:
  - o strontium ranelate
  - o raloxifene
  - o teriparatide
  - romosozumab

# **Clinical scenarios**

Fragility risk fracture assessment

- if a patient is deemed high-risk based on a QFracture or FRAX score they should have a DEXA scan to assess bone mineral density (BMD)
  - $\circ~$  the BMD threshold for defining osteoporosis is a T-score of 2.5 SD or below
  - some patients may not be suitable for BMD assessment due to frailty etc.
- general osteoporosis management as above
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

Postmenopausal women, and men age ≥50, who are treated with oral glucocorticoids:

- if starting ≥7.5 mg/day prednisolone or equivalent for the next 3 months, start bone protective treatment at the same time
- general osteoporosis management as above
- don't wait for a DEXA scan before starting treatment
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

A postmenopausal woman, or a man age ≥50 has a symptomatic osteoporotic vertebral fracture:

- general osteoporosis management as above
- start treatment straight away oral bisphosphonates are used first-line e.g. alendronate or risedronate

#### Hip fracture in older adults

- in older adults a hip fracture is a manifestation of osteoporosis
  - o following a fragility fracture in women ≥ 75 years, a DEXA scan is not necessary to diagnose osteoporosis and hence commence a bisphosphonate
  - BMD should be measured, but this acts as a baseline rather than determining whether treatment should be given
- bisphosphonates should be given first-line
  - NOGG recommends IV zoledronate but local guidelines may vary and oral bisphosphonates are often used

# Follow-up

Plan to prescribe oral bisphosphonates for at least 5 years, or intravenous bisphosphonates for at least 3 years and then re-assess fracture risk.

# Supplementary notes on treatment

## Bisphosphonates

- bisphosphonates bind to hydroxyapatite in bone, inhibiting osteoclast-mediated bone resorption
- common side effects include gastrointestinal discomfort, oesophagitis, and hypocalcaemia. Atypical femoral fractures and osteonecrosis of the jaw are rare but serious risks.
- available in oral and intravenous formulations. Oral bisphosphonates should be taken with a full glass of water, on an empty stomach, and the patient should remain upright for at least 30 minutes afterwards.

#### Denosumab

- human monoclonal antibody that inhibits RANK ligand, which in turn inhibits the maturation of osteoclasts
- also used for cancer patients with bone metastases to reduce skeletal-related events.
- given as a single subcutaneous injection every 6 months

## Raloxifene

- selective oestrogen receptor modulator (SERM)
- has been shown to prevent bone loss and to reduce the risk of vertebral fractures, but has not yet been shown to reduce the risk of non-vertebral fractures
- has been shown to increase bone density in the spine and proximal femur
- may worsen menopausal symptoms
- increased risk of thromboembolic events

• may decrease the risk of breast cancer

#### Strontium ranelate

- 'dual action bone agent' increases deposition of new bone by osteoblasts (promotes differentiation of pre-osteoblast to osteoblast) and reduces the resorption of bone by inhibiting osteoclasts
- concerns regarding the safety profile of strontium have been raised recently. It should only be prescribed by a specialist in secondary care
- due to these concerns the European Medicines Agency in 2014 said it should only be used by people for whom there are no other treatments for osteoporosis
- increased risk of cardiovascular events: any history of cardiovascular disease or significant risk of cardiovascular disease is a contraindication
- increased risk of thromboembolic events: a Drug Safety Update in 2012 recommended it is not used in patients with a history of venous thromboembolism
- may cause serious skin reactions such as Stevens Johnson syndrome

#### Teriparatide

- recombinant form of parathyroid hormone
- very effective at increasing bone mineral density but role in the management of osteoporosis yet to be clearly defined

#### Romosozumab

- a monoclonal antibody that inhibits sclerostin, thereby increasing bone formation and decreasing bone resorption
- this dual action significantly improves bone density and reduces fracture risk.



© Image used on license from Radiopaedia

MRI showing osteoporotic fractures of the 10th and 12th thoracic vertebrae.



Next question >

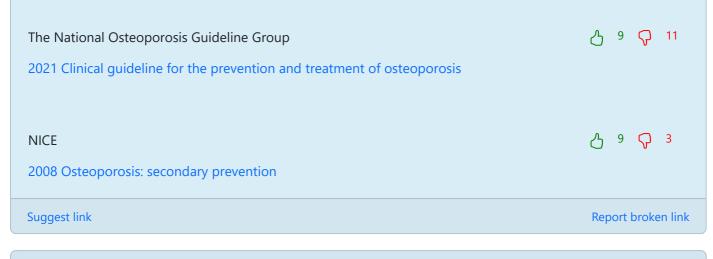


# **Textbooks**

High-yield textbook

Extended textbook

# Links



## Media



Osteoporosis pharmacology, prevention and treatment

Armando Hasudungan - YouTube







Report broken media

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A 77-year-old lady is reviewed in the Rheumatology Clinic with a 4-week history of malaise and bilateral hip pain.

The pain is poorly localised and affects the anterior and posterior aspects of the pelvis as well as the upper thighs. It is typically worse in the mornings and associated with feelings of stiffness that take several hours to improve.

She also reports the recent onset of a right-sided headache, which is constant and has been present for the past 2 weeks. 24 hours ago, she developed an episode of transient visual darkening although she is unable to recall which eye was affected.

Her past medical history is remarkable for hypertension and hypothyroidism. Her regular medications include amlodipine 5mg once daily and levothyroxine 75 micrograms once daily.

On examination, her visual acuity is 6/9 in both eyes. Her temperature is 37.3°C, her pulse is 73bpm and her blood pressure is 143/81mmHg. Neurological examination reveals no focal abnormality although the pulsation of her right temporal artery is difficult to feel.

Her blood results are as follows:

Hb	124 g/l	Na <sup>+</sup>	141 mmol/l
Platelets	444 * 10 <sup>9</sup> /l	K <sup>+</sup>	3.9 mmol/l
WBC	11.2 * 10 <sup>9</sup> /l	Urea	4.3 mmol/l
Neuts	8.1 * 10 <sup>9</sup> /l	Creatinine	78 µmol/l
Lymphs	2.3 * 10 <sup>9</sup> /l	CRP	102 mg/l
Eosin	0.02 * 10 <sup>9</sup> /l		

A temporal artery biopsy is performed and a 0.8cm sample is obtained. It is reported as being 'negative for giant cell arteritis (GCA)'.

What is the most appropriate treatment strategy?

IV methylprednisolone 1 gram once daily	
Prednisolone 15mg once daily	
Amitriptyline 10mg once daily	

Explanation, reassurance and referral to physiotherapy for graded aerobic exercise	
Prednisolone 60mg once daily	

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Question 68 of 178



 $\Box$ 



A 77-year-old lady is reviewed in the Rheumatology Clinic with a 4-week history of malaise and bilateral hip pain.

The pain is poorly localised and affects the anterior and posterior aspects of the pelvis as well as the upper thighs. It is typically worse in the mornings and associated with feelings of stiffness that take several hours to improve.

She also reports the recent onset of a right-sided headache, which is constant and has been present for the past 2 weeks. 24 hours ago, she developed an episode of transient visual darkening although she is unable to recall which eye was affected.

Her past medical history is remarkable for hypertension and hypothyroidism. Her regular medications include amlodipine 5mg once daily and levothyroxine 75 micrograms once daily.

On examination, her visual acuity is 6/9 in both eyes. Her temperature is 37.3°C, her pulse is 73bpm and her blood pressure is 143/81mmHg. Neurological examination reveals no focal abnormality although the pulsation of her right temporal artery is difficult to feel.

Her blood results are as follows:

Hb	124 g/l	Na <sup>+</sup>	141 mmol/l
Platelets	444 * 10 <sup>9</sup> /l	K <sup>+</sup>	3.9 mmol/l
WBC	11.2 * 10 <sup>9</sup> /l	Urea	4.3 mmol/l
Neuts	8.1 * 10 <sup>9</sup> /l	Creatinine	78 µmol/l
Lymphs	2.3 * 10 <sup>9</sup> /l	CRP	102 mg/l
Eosin	0.02 * 10 <sup>9</sup> /l		

A temporal artery biopsy is performed and a 0.8cm sample is obtained. It is reported as being 'negative for giant cell arteritis (GCA)'.

What is the most appropriate treatment strategy?





## Prednisolone 60mg once daily

34%

This lady's presentation is consistent with GCA. The picture is complicated by additional features of polymyalgia rhuematica (PMR) although it is important to bear in mind that the two often occur together in clinical practice.

A normal temporal artery biopsy does not rule out GCA due to the possibility of skip lesions giving rise to a false negative result. 7-44% of patients with GCA will have a negative temporal artery biopsy and such falsely reassuring results are more likely to occur when shorter arterial specimens are obtained. For this reason, the British Society for Rheumatology (BSR) recommends that biopsy specimens should no less than 1cm in length.

Transient visual loss can herald the onset of permanent blindness and the BSR recommend that these patients receive IV methylprednisolone 500-1000mg daily for 3 days.

Those with established visual loss or uncomplicated GCA should receive 60mg prednisolone daily.

Prednisolone 15mg daily is the treatment for isolated PMR and would not be appropriate in this case. The remaining options are treatments for fibromyalgia and are therefore incorrect.



Next question >

# Temporal arteritis \*

Temporal arteritis (also known as giant cell arteritis: GCA) is a vasculitis of unknown cause that affects medium and large-sized vessels arteries. It occurs in those over 50 years old, with a peak incidence in patients who are in their 70s.

It requires early recognition and treatment to minimize the risk of complications such as permanent loss of vision. Hence, when temporal arteritis is suspected, treatment must be started promptly with high-dose prednisolone as well as urgent referral for assessment by a specialist.

There is an overlap between temporal arteritis and polymyalgia rheumatica (PMR) - around 50% of patients will have features of PMR.

#### **Features**

- typically patient > 60 years old
- usually rapid onset (e.g. < 1 month)</li>
- headache (found in 85%)

- jaw claudication (65%)
- vision testing is a key investigation in all patients
  - anterior ischemic optic neuropathy accounts for the majority of ocular complications. It results from occlusion of the posterior ciliary artery (a branch of the ophthalmic artery) → ischaemia of the optic nerve head. Fundoscopy typically shows a swollen pale disc and blurred margins
  - o may result in temporary visual loss amaurosis fugax
  - permanent visual loss is the most feared complication of temporal arteritis and may develop suddenly
  - diplopia may also result from the involvement of any part of the oculomotor system (e.g. cranial nerves)
- tender, palpable temporal artery
- around 50% have features of PMR: aching, morning stiffness in proximal limb muscles (not weakness)
- also lethargy, depression, low-grade fever, anorexia, night sweats

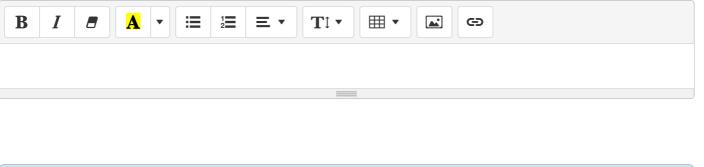
#### Investigations

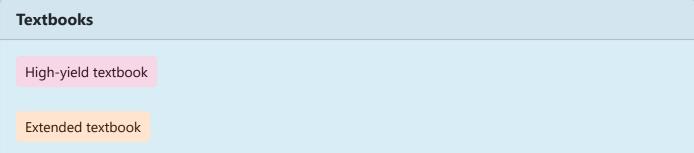
- raised inflammatory markers
  - ESR > 50 mm/hr (note ESR < 30 in 10% of patients)
  - CRP may also be elevated
- temporal artery biopsy
  - o skip lesions may be present
- note creatine kinase and EMG normal

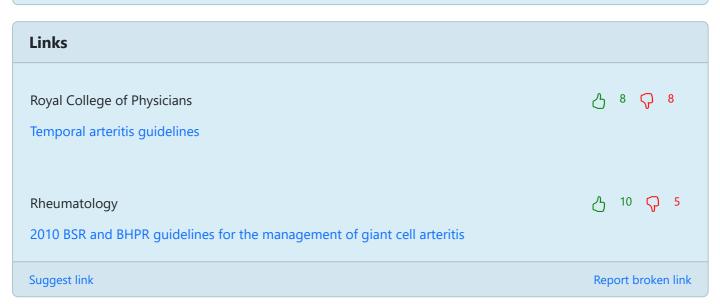
#### **Treatment**

- urgent high-dose glucocorticoids should be given as soon as the diagnosis is suspected and before the temporal artery biopsy
  - o if there is no visual loss then high-dose prednisolone is used
  - if there is evolving visual loss IV methylprednisolone is usually given prior to starting high-dose prednisolone
  - there should be a dramatic response, if not the diagnosis should be reconsidered
- urgent ophthalmology review
  - o patients with visual symptoms should be seen the same-day by an ophthalmologist
  - visual damage is often irreversible
- other treatments
  - bone protection with bisphosphonates is required as long, tapering course of steroids is required
  - low-dose aspirin is sometimes given to patients as well, although the evidence base supporting this is weak













## **Temporal Arteritis**

Pixorize - YouTube









#### Temporal arteritis

Khan Academy - YouTube









## Temporal artery biopsy

Oculoplastics.info - YouTube







Giant cell Arteritis and Takayasu arteritis (Large Vessel Vasculitis) - signs, pathophysiology

Armando Hasudungan - YouTube





Report broken media

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Question 69 of 178





A 28-year-old woman presents to the gastroenterology clinic for review. She has been diagnosed with coeliac disease some 2 years earlier, and has been suffering from severe tiredness, muscle aches and proximal weakness for the past few months. On examination her blood pressure is 112/70 mmHg, pulse is 75 beats per minute and regular. You confirm proximal muscle weakness.

## Investigations

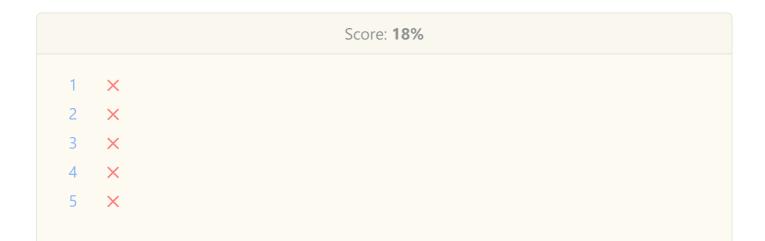
Ca++	2.0 mmol/l
Alkaline phosphatase	275 IU/I

Which of the following is the most useful next investigation?

CK	
Parathyroid hormone	
Vitamin D	
Muscle biopsy	
Electromyography	

Submit answer

Reference ranges ∨



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Question 69 of 178



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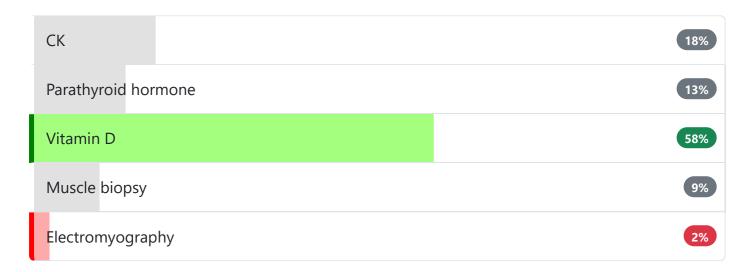


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#### Investigations

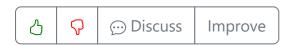
Ca <sup>++</sup>	2.0 mmol/l
Alkaline phosphatase	275 IU/I

Which of the following is the most useful next investigation?



Coeliac disease is known to interfere with absorption of fat soluble vitamins, including vitamin D, and the low calcium and elevated alkaline phosphatase, coupled with symptoms of proximal myopathy fits with a diagnosis of osteomalacia. Vitamin D levels are therefore the next investigation of choice.

CK and muscle biopsy are indicated for possible inflammatory myositis, and the limited proximal weakness seen here, coupled with low calcium is much more consistent with osteomalacia. Parathyroid hormone may be elevated, but this is secondary to low vitamin D and calcium. Electromyography is most useful for assessment of motor neuropathy, which doesn't fit with the painful proximal muscle weakness seen here.



# Osteomalacia \*

Osteomalacia describes softening of the bones secondary to low vitamin D levels that in turn lead to decreased bone mineral content. If this occurs in growing children it is referred to as rickets, with the term osteomalacia preferred for adults.

#### Causes

- vitamin D deficiency
  - o malabsorption
  - o lack of sunlight
  - o diet
- chronic kidney disease
- drug induced e.g. anticonvulsants
- inherited: hypophosphatemic rickets (previously called vitamin D-resistant rickets)
- liver disease: e.g. cirrhosis
- coeliac disease

#### **Features**

- bone pain
- bone/muscle tenderness
- fractures: especially femoral neck
- proximal myopathy: may lead to a waddling gait

#### Investigation

- bloods
  - low vitamin D levels
  - low calcium, phosphate (in around 30%)
  - raised alkaline phosphatase (in 95-100% of patients)
- x-ray
  - translucent bands (Looser's zones or pseudofractures)

#### **Treatment**

- vitamin D supplmentation
  - a loading dose is often needed initially
- calcium supplementation if dietary calcium is inadequate

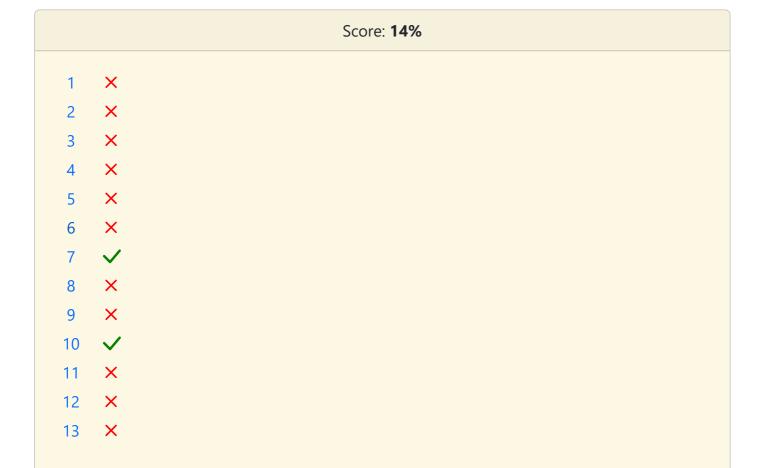


Next question >



# Textbooks High-yield textbook Extended textbook





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A 30-year-old man presents to the emergency department with worsening neck pain, which limits his participation in contact sports. He is generally well otherwise and does not take any regular medications. He smokes ten cigarettes daily and drinks alcohol occasionally.

The plain film radiograph of his C-spine showed:



What is the next best step in his management?

Commence etanercept	
Commence regular naproxen	
Commence sulfasalazine	
Limit physical exercise	
Urgent referral to spine surgery	

# Submit answer

Reference ranges  $\checkmark$ 

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## Score: **18%** X 1 2 X 3 X 4 × 5 X 6 X 7 X 8 9 X 10 X 11 X 12 X 13 14 X 15 **/** 16 × 17 X 18 X 19 × 20 21 X 22 X 23 X × 24 25 26 × 27 X 28

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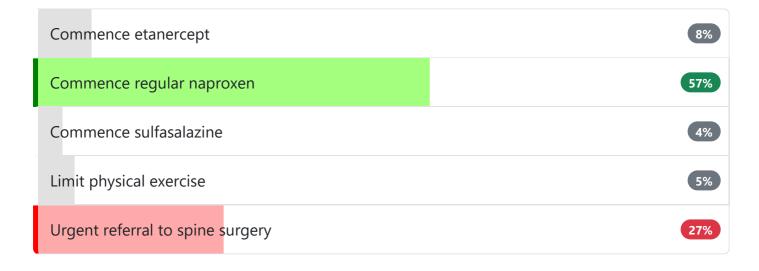


A 30-year-old man presents to the emergency department with worsening neck pain, which limits his participation in contact sports. He is generally well otherwise and does not take any regular medications. He smokes ten cigarettes daily and drinks alcohol occasionally.

The plain film radiograph of his C-spine showed:



What is the next best step in his management?



The neck radiograph shows syndesmophytes, squaring of the vertebrae and bamboo spine, characteristic of ankylosing spondylitis.

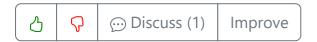
**Commence regular naproxen** is the correct answer. NSAIDs are the first-line treatment for ankylosing spondylitis. Therefore the patient should be started on oral NSAIDs first.

**Commence etanercept** is incorrect. The 2010 European Alliance of Associations for Rheumatology (EULAR) guidelines suggest: 'anti-TNF therapy should be given to patients with persistently high disease activity despite conventional treatments'.

**Commence sulfasalazine** is incorrect. Disease-modifying drugs used to treat rheumatoid arthritis (such as sulphasalazine) are only useful in ankylosing spondylitis if peripheral joint involvement exists.

**Limit physical exercise** is incorrect. Physiotherapy and regular exercise are recommended treatments for ankylosing spondylitis as per the EULAR guidelines.

**Urgent referral to spine surgery** is incorrect. The most common surgical procedure for ankylosing spondylitis is a laminectomy to relieve pressure on the nerve roots. However, as this patient has no features of nerve root compression, medical therapy is the next best step in his management.



Next question >

# Ankylosing spondylitis: investigation and management \*



Ankylosing spondylitis is a HLA-B27 associated spondyloarthropathy. It typically presents in males (sex ratio 3:1) aged 20-30 years old.

# Investigation

Inflammatory markers (ESR, CRP) are typically raised although normal levels do not exclude ankylosing spondylitis.

HLA-B27 is of little use in making the diagnosis as it is positive in:

- 90% of patients with ankylosing spondylitis
- 10% of normal patients

Plain x-ray of the sacroiliac joints is the most useful investigation in establishing the diagnosis. Radiographs may be normal early in disease, later changes include:

- sacroiliitis: subchondral erosions, sclerosis
- squaring of lumbar vertebrae
- 'bamboo spine' (late & uncommon)
- syndesmophytes: due to ossification of outer fibers of annulus fibrosus
- chest x-ray: apical fibrosis

If the x-ray is negative for sacroiliac joint involvement in ankylosing spondylitis but suspicion for AS remains high, the next step in the evaluation should be obtaining an MRI. Signs of early inflammation involving sacroiliac joints (bone marrow oedema) confirm the diagnosis of AS and prompt further treatment.

Spirometry may show a restrictive defect due to a combination of pulmonary fibrosis, kyphosis and ankylosis of the costovertebral joints.













## Management

The following is partly based on the 2010 EULAR guidelines (please see the link for more details):

- encourage regular exercise such as swimming
- NSAIDs are the first-line treatment
- physiotherapy
- the disease-modifying drugs which are used to treat rheumatoid arthritis (such as sulphasalazine) are only really useful if there is peripheral joint involvement
- the 2010 EULAR guidelines suggest: 'Anti-TNF therapy should be given to patients with persistently high disease activity despite conventional treatments'
- research is ongoing to see whether anti-TNF therapies such as etanercept and adalimumab should be used earlier in the course of the disease

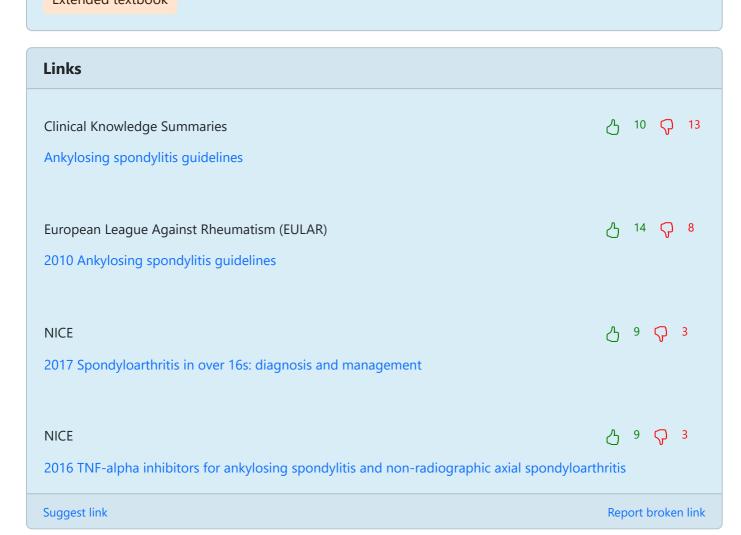
















Ankylosing spondylitis

Zero to Finals - YouTube







## Ankylosing spondylitis

Townsend Teaching - YouTube 6 1 1 1



## Ankylosing spondylitis

Osmosis - YouTube



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Question 71 of 178





A 53-year-old has just been diagnosed with rheumatoid arthritis whilst having a severe flare. She is started on methotrexate 15mg once weekly, folic acid 5mg once weekly, hydroxychloroquine 200mg BD, naproxen 250mg TDS and prednisolone 15mg OD. She returns one month later complaining of mouth ulcers. Bloods show the following:

Hb	142 g/l
Platelets	225 * 10 <sup>9</sup> /l
WBC	6 * 10 <sup>9</sup> /l
Na <sup>+</sup>	136 mmol/l
K <sup>+</sup>	4.2 mmol/l
Urea	4 mmol/l
Creatinine	95 µmol/l
Bilirubin	6 µmol/l
ALP	105 u/l
ALT	92 u/l

What is the most appropriate course of action?

Admit for IV methylprednisolone	
Stop methotrexate, hydroxychloroquine and naproxen	
Increase folic acid to two days a week	
Stop hydroxychloroquine and discuss with rheumatology	
Stop methotrexate and discuss with rheumatology	

Submit answer

Reference ranges ∨

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Question 71 of 178



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K <sup>+</sup>	4.2 mmol/l
Urea	4 mmol/l
Creatinine	95 µmol/l
Bilirubin	6 µmol/l
ALP	105 u/l
ALT	92 u/l

What is the most appropriate course of action?

Admit for IV methylprednisolone	6%
Stop methotrexate, hydroxychloroquine and naproxen	2%
Increase folic acid to two days a week	20%
Stop hydroxychloroquine and discuss with rheumatology	17%
Stop methotrexate and discuss with rheumatology	55%

According to BSR guidelines, if new oral ulceration starts whilst a patient is on methotrexate then it should be withheld initially and discussed with the specialist team.

In this patient, the alanine transaminase (ALT) is not two times the upper range of normal so that does not affect your decision. Often the folic acid is increased to six days a week (apart from the day of methotrexate) to mitigate side effects.

Hydroxychloroquine and naproxen are not associated with oral ulceration.

Next question >

## Methotrexate \*

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

#### Indications

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

#### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - o the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - typically develops within a year of starting treatment, either acutely or subacutely
  - o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

### Prescribing methotrexate

- methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose

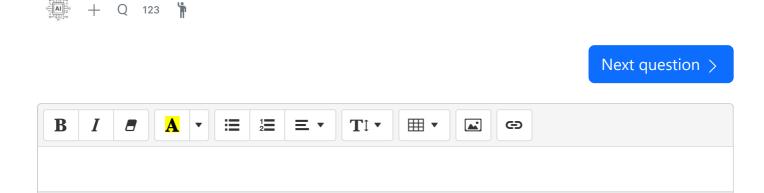
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

### Interactions

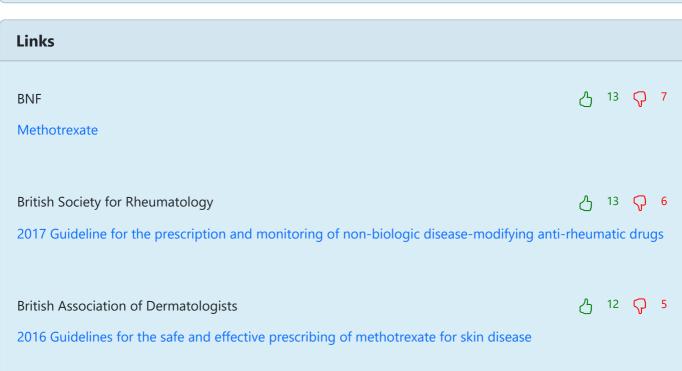
- avoid prescribing <u>trimethoprim</u> or <u>co-trimoxazole</u> concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

## Methotrexate toxicity

• the treatment of choice is folinic acid







Suggest link Report broken link

## Media



Methotrexate - Pharmacology (DMARDs, mechanism of action, side effects)

Armando Hasudungan - YouTube





Report broken media

Score: 14%

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A 60 year old woman attended her General Practitioner and reported a three month history of bilateral shoulder muscle and bilateral hip girdle aches and pain. She also experienced stiffness affecting these areas that lasted for up to two hours each morning. These symptoms were limiting her day to day activities and were unresponsive to simple analgesics.

The patient denied symptoms of headache, visual disturbance or jaw claudication. Intermittent episodes of dry mouth and dry eyes had been present for several years. There was no history of unexplained skin rashes. Past medical history included coeliac disease diagnosed twenty years previously that was well controlled on a gluten-free diet. The patient was a non-smoker and drank alcohol only occasionally.

Examination revealed mild muscular tenderness across the shoulder and hip girdles although with no other inflamed or tender joints. Cardiovascular and respiratory examination was unremarkable.

Blood tests requested by her GP demonstrated an elevated ESR of 65. A diagnosis of PMR was made and a course of 20 mg prednisolone daily prescribed. However 6 weeks later the patients symptoms had not significantly improved and she was referred to rheumatology clinic. Repeat blood tests and other investigations are listed below.

Haemoglobin	110 g / dL
White cell count	8.9 * 10 <sup>9</sup> /l
Neutrophils	7.8 * 10 <sup>9</sup> /l
Platelets	456 * 10 <sup>9</sup> /I
Urea	6.2 mmol / L
Creatinine	87 micromol / L
Sodium	138 mmol / L
Potassium	4.1 mmol / L
Ferritin	180 ng / mL
Erythrocyte sedimentation rate	75 mm / h
Rheumatoid factor	Negative
Connective tissue ANA	Negative
Anti-CCP antibodies	58 EU (reference < 20)
Creatinine kinase	89 U / L (reference 5-130)

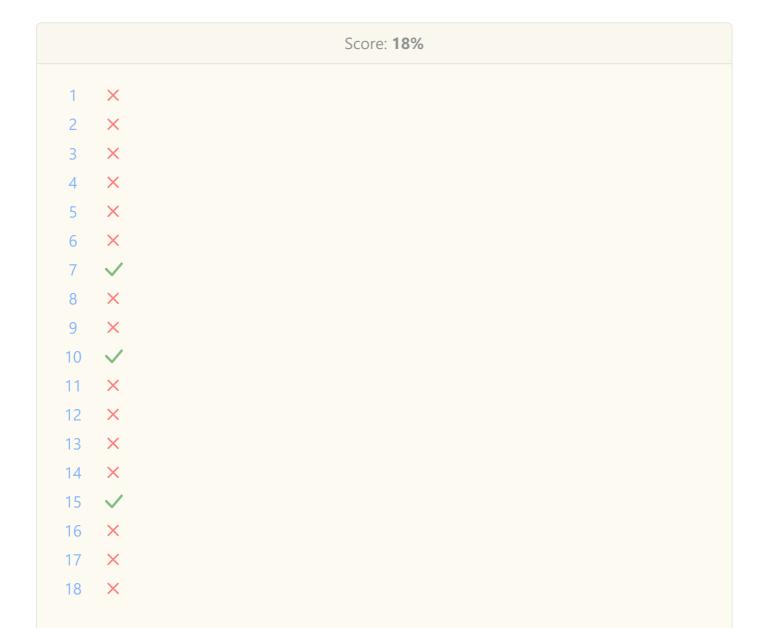
X-ray hands: minor degenerative change in multiple inter-phalangeal joints of both hands; no evidence of erosive arthropathy

# What is correct diagnosis?

Polymyalgia rheumatica	
Rheumatoid arthritis	
Polymyositis	
Sjorgren's syndrome	
Systemic lupus erythematous	

# Submit answer

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Question 72 of 178



 $\Box$ 



A 60 year old woman attended her General Practitioner and reported a three month history of bilateral shoulder muscle and bilateral hip girdle aches and pain. She also experienced stiffness affecting these areas that lasted for up to two hours each morning. These symptoms were limiting her day to day activities and were unresponsive to simple analgesics.

The patient denied symptoms of headache, visual disturbance or jaw claudication. Intermittent episodes of dry mouth and dry eyes had been present for several years. There was no history of unexplained skin rashes. Past medical history included coeliac disease diagnosed twenty years previously that was well controlled on a gluten-free diet. The patient was a non-smoker and drank alcohol only occasionally.

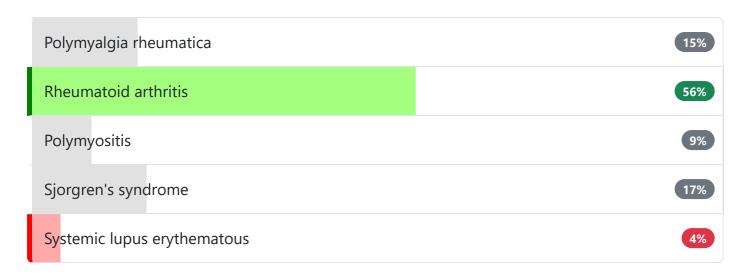
Examination revealed mild muscular tenderness across the shoulder and hip girdles although with no other inflamed or tender joints. Cardiovascular and respiratory examination was unremarkable.

Blood tests requested by her GP demonstrated an elevated ESR of 65. A diagnosis of PMR was made and a course of 20 mg prednisolone daily prescribed. However 6 weeks later the patients symptoms had not significantly improved and she was referred to rheumatology clinic. Repeat blood tests and other investigations are listed below.

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Urea	6.2 mmol / L
Creatinine	87 micromol / L
Sodium	138 mmol / L
Potassium	4.1 mmol / L
Ferritin	180 ng / mL
Erythrocyte sedimentation rate	75 mm / h
Rheumatoid factor	Negative
Connective tissue ANA	Negative
Anti-CCP antibodies	58 EU (reference < 20)
Creatinine kinase	89 U / L (reference 5-130)

X-ray hands: minor degenerative change in multiple inter-phalangeal joints of both hands; no evidence of erosive arthropathy

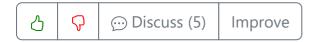
What is correct diagnosis?



Rheumatoid arthritis can present with a polymyalgic syndrome prior to clinically detectable sinovitis. In this case this is suggested by the lack of response to trial of prednisolone and the positive anti-CCP antibody. Observational studies have shown a greater clinical and laboratory response to steroids in polymyalgia rheumatica than polymyalgic onset rheumatoid arthritis. Anti-CCP antibodies are rarely present in polymyalgia rheumatica but are strongly associated with rheumatoid arthritis.

Sjorgren's syndrome and SLE are unlikely given the lack of anti-nuclear antibodies. Polymyositis is excluded by the normal CK.

Mackie S, Mallen C. Polymyalgia rheumatica. BMJ 2013;347:f6937.



Next question >

# Rheumatoid arthritis: diagnosis \*

NICE have stated that clinical diagnosis is more important than criteria such as those defined by the American College of Rheumatology.

## 2010 American College of Rheumatology criteria

Target population. Patients who

- 1) have at least 1 joint with definite clinical synovitis
- 2) with the synovitis not better explained by another disease

Classification criteria for rheumatoid arthritis (add score of categories A-D;

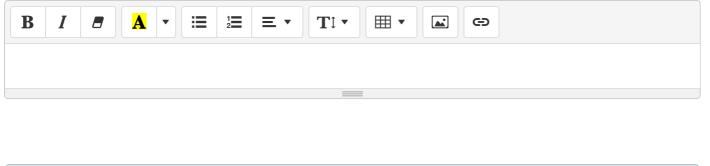
a score of 6/10 is needed definite rheumatoid arthritis)

## Key

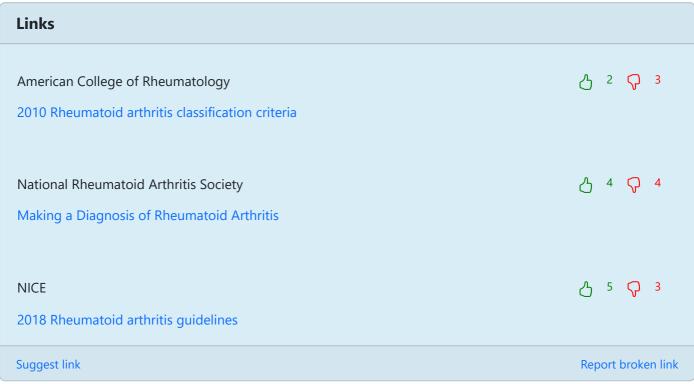
- RF = rheumatoid factor
- ACPA = anti-cyclic citrullinated peptide antibody

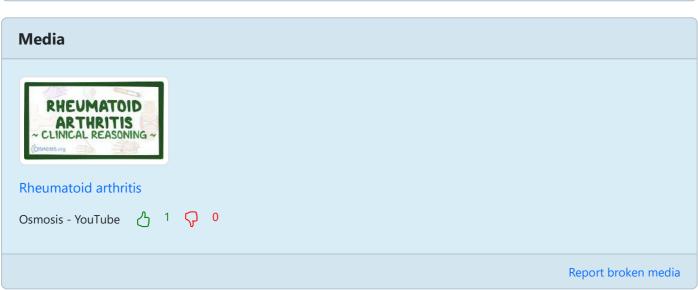
Factor	Scoring	
A. Joint involvement		
	1 large joint	0
	2 - 10 large joints	1
	1 - 3 small joints (with or without involvement of large joints)	2
	4 - 10 small joints (with or without involvement of large joints)	3
	10 joints (at least 1 small joint)	5
B. Serology (at least 1 test result is needed for classification)		
	Negative RF and negative ACPA	0
	Low-positive RF or low-positive ACPA	2
	High-positive RF or high-positive ACPA	3
C. Acute-phase reactants (at least 1 test result is needed for classification)		
	Normal CRP and normal ESR	0
	Abnormal CRP or abnormal ESR	1
D. Duration of symptoms		
	< 6 weeks	0
	> 6 weeks	1











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Question 73 of 178

P



A 65-year-old man presented to his General Practitioner with a 3-month history of bilateral shoulder aches and pains. The symptoms were associated with stiffness in the mornings taking up to two hours to resolve after waking. The patient denied any symptoms of headache, jaw claudication or visual disturbance. The patient had no symptoms of dry eyes or mouth, no skin or hair changes, no weight loss and no fevers.

Past medical history included hypertension and chronic obstructive pulmonary disease. Regular medications included ramipril, simvastatin and inhaled salbutamol as required. The patient was an ex-smoker who drank 25 units of alcohol per week. The patient had recently retired having spent his working life as a train driver.

The examination did not reveal any inflamed joints excepting slight tenderness across the shoulder girdle. There was no evidence of scalp tenderness. The cardiovascular and respiratory examination was unremarkable.

Investigations requested by the General Practitioner are listed below.

Haemoglobin	134 g / L
White cell count	7.5* 10 <sup>9</sup> /l
Neutrophils	6.0 * 10 <sup>9</sup> /l
Platelets	356 * 10 <sup>9</sup> /l
Urea	8.9 mmol / L
Creatinine	110 micromol / L
Sodium	132 mmol / L
Potassium	4.9 mmol / L
Erythrocyte sedimentation rate	85 mm / h
Rheumatoid factor	Negative
Creatinine kinase	121 U / L (reference 5-130)
Calcium (adjusted)	2.25 mmol / L (reference 2.18-2.58)
Alkaline phosphatase	67 U / L (reference 35-100)
Thyroid stimulating hormone	2.5 microU / L
Protein electrophoresis	Normal

What is the appropriate next management step for this patient?

Stop statin therapy and review in 6 weeks	
Ultrasound study of shoulders and hips	×
Referral for specialist rheumatology opinion	×
Prednisolone 15 mg daily with dose tapering over 2 years	×
Prednisolone 40 mg daily with dose tapering over 1 year	

# Submit answer

Reference ranges  $\vee$ 

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Question 73 of 178



 $\square$ 



A 65-year-old man presented to his General Practitioner with a 3-month history of bilateral shoulder aches and pains. The symptoms were associated with stiffness in the mornings taking up to two hours to resolve after waking. The patient denied any symptoms of headache, jaw claudication or visual disturbance. The patient had no symptoms of dry eyes or mouth, no skin or hair changes, no weight loss and no fevers.

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Alkaline phosphatase	67 U / L (reference 35-100)
Thyroid stimulating hormone	2.5 microU / L
Protein electrophoresis	Normal

What is the appropriate next management step for this patient?

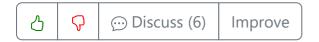
Stop statin therapy and review in 6 weeks	12%
Ultrasound study of shoulders and hips	5%
Referral for specialist rheumatology opinion	15%
Prednisolone 15 mg daily with dose tapering over 2 years	47%
Prednisolone 40 mg daily with dose tapering over 1 year	21%

This patient presents with a classical history for polymyalgia rheumatica and a raised ESR. There are no factors in the history or investigations that suggest an alternative diagnosis (for example, giant cell arteritis, other connective tissue disease, myeloma, malignancy or occult infection). The normal CK would make statin-induced myopathy unlikely.

In such cases, a trial of steroid therapy for likely polymyalgia rheumatica is appropriate. The diagnosis will be confirmed by rapid resolution of symptoms following initiation of treatment. Observational studies suggest a typical starting dose of prednisolone around 15 mg with a median time to stopping therapy of two years.

Musculoskeletal ultrasound often identifies inflammation around the shoulders and hips although these findings are not unique to polymyalgia rheumatica. The usefulness of this technique is yet to be determined outside of specialist settings.

Mackie S, Mallen C. Polymyalgia rheumatica. BMJ 2013;347:f6937



Next question >

# Polymyalgia rheumatica \*

Polymyalgia rheumatica (PMR) is a relatively common condition seen in older people characterised by muscle stiffness and raised inflammatory markers. Whilst it appears to be closely related to temporal arteritis the underlying cause is not fully understood and it does not appear to be a vasculitic process.

#### **Features**

- typically patient > 60 years old
- usually rapid onset (e.g. < 1 month)</li>
- aching, morning stiffness in proximal limb muscles
  - weakness is not considered a symptom of polymyalgia rheumatica

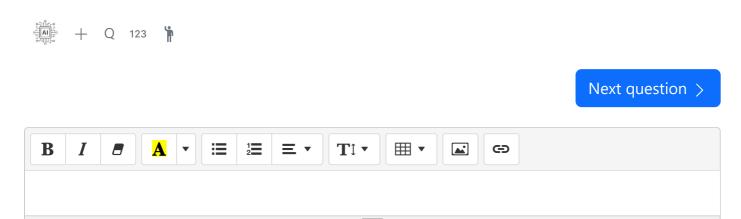
• also mild polyarthralgia, lethargy, depression, low-grade fever, anorexia, night sweats

## Investigations

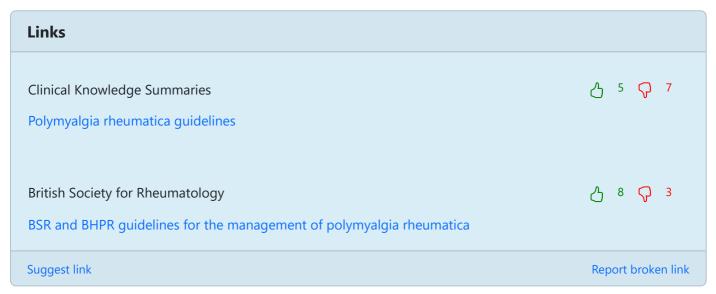
- raised inflammatory markers e.g. ESR > 40 mm/hr
- note creatine kinase and EMG normal

### **Treatment**

- prednisolone e.g. 15mg/od
  - patients typically respond dramatically to steroids, failure to do so should prompt consideration of an alternative diagnosis







## Media



## Polymyalgia rheumatica

Zero To Finals - YouTube









### Polymyalgia rheumatica

Osmosis - YouTube











## Polymyalgia rheumatica

Townsend Teaching - YouTube  $\bigcirc$  0  $\bigcirc$  0







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A 40-year-old woman was diagnosed with fibromyalgia 6 months previously following review in the Rheumatology outpatient clinic. She was subsequently discharged from the clinic with a recommendation for a trial of pregabalin therapy. The patient has now returned to her General Practitioner to report on-going symptoms of severe widespread body pain, severe fatigue and difficulty in concentrating on daily activities. The patient did not feel that starting pregabalin 6 months previously had offered any improvement in her symptoms. In fact, her symptoms had been causing more problems and she had recently been unable to attend her work as a teaching assistant.

The patients past medical history included a duodenal ulcer five years previously, induced by the combination of non-steroidal anti-inflammatory drug use and alcohol consumption. She also had a previous diagnosis of irritable bowel disease and had a tendency to become severely constipated. There were no known allergies to medications. The patient lived alone and in addition to her teaching assistant job was her elderly mother's primary carer.

On assessment by her General Practitioner, there was no evidence of inflammatory arthritis but multiple tender spots were demonstrated across the patient's body. The patient was clearly distressed and frustrated with her on-going symptoms and had concerns about her ability to continue in paid employment. The patients affect was otherwise unremarkable with a good rapport maintained throughout. She denied any symptoms of low mood or thoughts of self-harm.

Following further discussion, the patient was keen to try a further pharmacological therapy as treatment for her fibromyalgia symptoms. She was reluctant to engage with suggested psychological therapies.

What is appropriate next line pharmacological treatment for the patient's fibromyalgia?

Continue pregabalin, start duloxetine	
Continue pregabalin, start ibuprofen as required	
Stop pregabalin, start duloxetine	
Stop pregabalin, start amitriptyline	
Continue pregabalin, start fluoxetine	

Submit answer

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Question 74 of 178



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Following further discussion, the patient was keen to try a further pharmacological therapy as treatment for her fibromyalgia symptoms. She was reluctant to engage with suggested psychological therapies.

What is appropriate next line pharmacological treatment for the patient's fibromyalgia?

Continue pregabalin, start duloxetine	10%
Continue pregabalin, start ibuprofen as required	2%
Stop pregabalin, start duloxetine	53%
Stop pregabalin, start amitriptyline	30%
Continue pregabalin, start fluoxetine	6%

The evidence base for pharmacological treatment of fibromyalgia includes many trials with small participant numbers and short follow-up periods. In addition, many trials have exclusion criteria (such as co-morbid psychiatric illness or chronic physical illness) that make generalisation to

patients seen in clinical practice difficult.

Recent network meta-analysis has demonstrated that once trials with fewer than 50 participants are excluded then there is only evidence of effectiveness of duloxetine and pregabalin to improve pain and quality of life. Current recommendation is to use an agent at an effective dose for at least four weeks then assess response. If no response obtained then initial treatment should be held before a new agent is started, for example stopping pregabalin before a trial of duloxetine as in this case.

Using ibuprofen or amitriptyline would likely be inappropriate for this patient given the previous history of peptic ulceration and constipation.

Carnes D, Underwood M, Rahman A. Fibromyalgia. BMJ 2014;348:g474.



Next question >

# Fibromyalgia 🖈

Fibromyalgia is a syndrome characterised by widespread pain throughout the body with tender points at specific anatomical sites. The cause of fibromyalgia is unknown.

## **Epidemiology**

- women are around 5 times more likely to be affected
- typically presents between 30-50 years old

#### **Features**

- chronic pain: at multiple site, sometimes 'pain all over'
- lethargy
- cognitive impairment: 'fibro fog'
- sleep disturbance, headaches, dizziness are common

Diagnosis is clinical and sometimes refers to the American College of Rheumatology classification criteria which lists 9 pairs of tender points on the body. If a patient is tender in at least 11 of these 18 points it makes a diagnosis of fibromyalgia more likely

The management of fibromyalgia is often difficult and needs to be tailored to the individual patient. A psychosocial and multidisciplinary approach is helpful. Unfortunately there is currently a paucity of evidence and guidelines to guide practice. The following is partly based on consensus guidelines from the European League against Rheumatism (EULAR) published in 2007 and also a BMJ review in 2014.

- explanation
- aerobic exercise: has the strongest evidence base
- cognitive behavioural therapy
- medication: pregabalin, duloxetine, amitriptyline



Next question >





High-yield textbook

Extended textbook

### Media



## Fibromyalgia

Townsend Teaching - YouTube 2 Q 1



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A 62-year-old man presents with a week of right-sided shoulder pain. He denies any trauma and denies radiation of the pain. There is no associated weakness or numbness. The pain is more noticeable at the extremities of movement and is affecting activities of daily living. His past medical history includes type 2 diabetes mellitus and asthma.

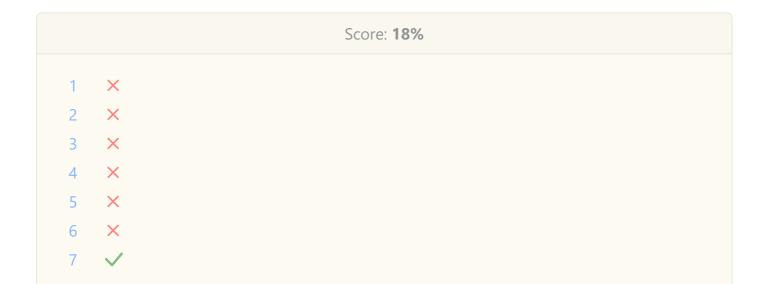
On examination, the affected shoulder is painful and restricted, with both active and passive movement. External rotation is most severely limited. Pain is elicited when applying direct pressure to the coracoid process.

Given the most likely diagnosis, what is the most appropriate next step?

Arthroscopy	
MRI scan	
Physiotherapy	
Ultrasound	
X-ray	

Submit answer

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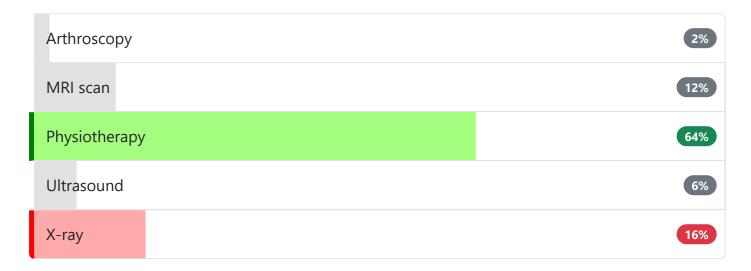
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A 62-year-old man presents with a week of right-sided shoulder pain. He denies any trauma and denies radiation of the pain. There is no associated weakness or numbness. The pain is more noticeable at the extremities of movement and is affecting activities of daily living. His past medical history includes type 2 diabetes mellitus and asthma.

On examination, the affected shoulder is painful and restricted, with both active and passive movement. External rotation is most severely limited. Pain is elicited when applying direct pressure to the coracoid process.

Given the most likely diagnosis, what is the most appropriate next step?



Adhesive capsulitis is a clinical diagnosis and does not need imaging or arthroscopy to confirm the diagnosis

Important for me Less important

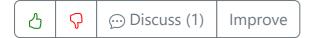
The diagnosis here is most likely to be adhesive capsulitis (frozen shoulder). This is evidenced by the reduction in movement, associated pain, and external rotation being more affected. The background of diabetes mellitus is also a risk factor. Adhesive capsulitis is generally a clinical diagnosis and so imaging/investigation is not required. **Physiotherapy** is therefore the correct answer and is the mainstay of treatment. It should be initiated as early on as possible.

**Arthroscopy** is inappropriate. This may be considered much further down the line as a therapeutic option if previous measures have failed to control the symptoms.

**MRI scanning** is unwarranted. This may be useful if the diagnosis were not as clear-cut and other pathologies needed to be ruled out.

**Ultrasound** may be considered before MRI if the diagnosis is uncertain and differentials need to be ruled out. However, in a case such as this, the clinical picture is sufficient to make the diagnosis.

An **X-ray** may be considered to exclude dislocations/fractures. However, in the history above, the picture is very strongly suggestive of adhesive capsulitis and so imaging is not needed. Physiotherapy should be initiated.



Next question >

# Adhesive capsulitis \*

Adhesive capsulitis (frozen shoulder) is a common cause of shoulder pain. It is most common in middle-aged females. The aetiology of frozen shoulder is not fully understood.

#### Associations

• diabetes mellitus: up to 20% of diabetics may have an episode of frozen shoulder

Features typically develop over days

- external rotation is affected more than internal rotation or abduction
- both active and passive movement is affected
- patients typically have a painful freezing phase, an adhesive phase and a recovery phase
- bilateral in up to 20% of patients
- the episode typically lasts between 6 months and 2 years

The diagnosis is usually clinical although imaging may be required for atypical or persistent symptoms.

## Management

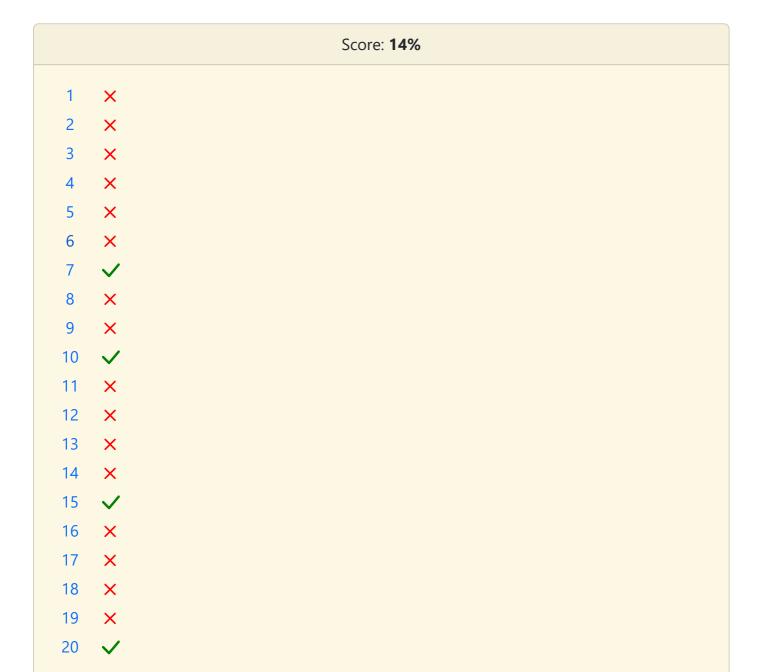
- no single intervention has been shown to improve outcome in the long-term
- treatment options include NSAIDs, physiotherapy, oral corticosteroids and intra-articular corticosteroids



Next question >







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Question 76 of 178





A 55-year-old female presents with increasing right-sided groin pain on activity. She recently joined a gym in an attempt to lose weight after being diagnosed with obesity. She has been started a number of activities including cycling, running and aerobic classes. The patient first noted the pain 3 months ago following a running session. She was able to continue with her exercise but the pain has become progressively worse and she now finds her gym sessions very difficult to complete.

Other than being overweight the patient has known hypothyroidism and asthma which both are well controlled on treatment.

An X-ray of the patient's hip is performed as below.



© Image used on license from Radiopaedia

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What is the patient's most likely diagnosis?

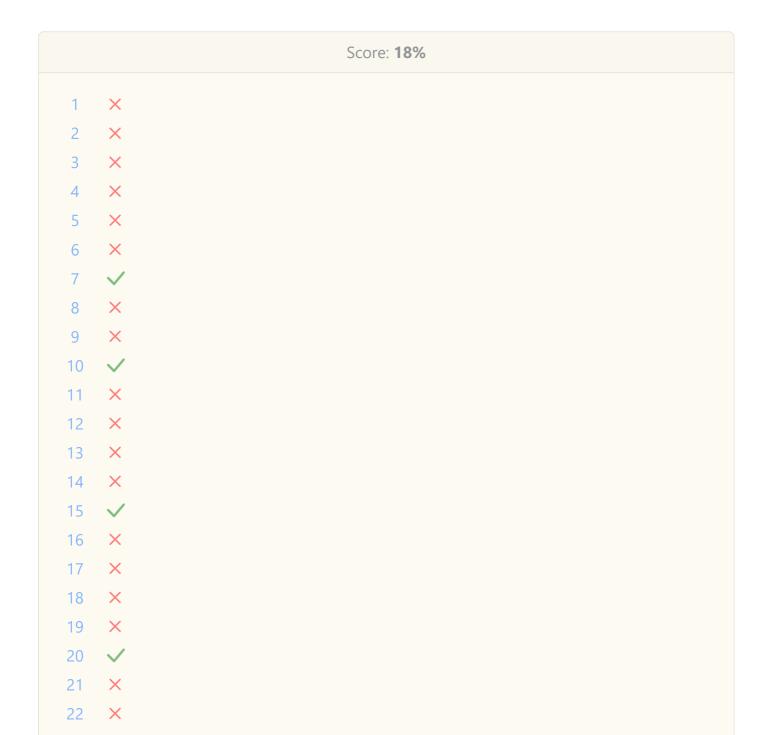


Avascular necrosis

Greater trochanteric pain syndrome (GTPS)	
Inflammatory arthritis	
Osteoarthritis	
Hip dysplasia	

## Submit answer

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Question 76 of 178

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 $\Box$ 



A 55-year-old female presents with increasing right-sided groin pain on activity. She recently joined a gym in an attempt to lose weight after being diagnosed with obesity. She has been started a number of activities including cycling, running and aerobic classes. The patient first noted the pain 3 months ago following a running session. She was able to continue with her exercise but the pain has become progressively worse and she now finds her gym sessions very difficult to complete.

Other than being overweight the patient has known hypothyroidism and asthma which both are well controlled on treatment.

An X-ray of the patient's hip is performed as below.



© Image used on license from Radiopaedia



What is the patient's most likely diagnosis?

Greater trochanteric pain syndrome (GTI	PS) 6%
Inflammatory arthritis	2%
Osteoarthritis	43%
Hip dysplasia	2%

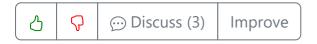
This patient has presented with the typical history and x-ray findings of osteoarthritis (OA). Risk factors for OA include obesity, increasing age and female gender. OA normally affects the knees and hip although most joints can be involved. When presenting with hip OA patients commonly report chronic, progressive groin pain on or post-exercise. Diagnosis is normal clinical and based on x-ray findings, as seen on this patient's imaging, which includes joint space loss, osteophyte formation, cyst formation and subchondral sclerosis.

Avascular necrosis (AVN) is bone breakdown due to a disruption of the blood supply. It is very rare in the absence of risk factors such as fracture, joint dislocations, alcoholism or high-dose steroids use. This patient does not have any clear risk factors of AVN, nor does she have any of the typical x-ray findings, which are normally only apparent in advanced disease, including bone flatting, subchondral radiolucent lines and collapsed femoral head.

Greater trochanteric pain syndrome (GTPS) is inflammation of the trochanteric bursa and is often a manifestation of an acute hip injury. Although this patient has the typical signs of pain on movement and activity, x-ray changes are very rare in GTPS.

Inflammatory arthritis includes conditions such as rheumatoid arthritis which are due to systemic disease. As such isolated joint involvement is rare especially of the hip with the condition commonly affecting the hands, feet and knees. X-ray findings include subchondral cysts and evidence of soft tissue swelling.

Hip dysplasia is an abnormality of the hip joint that normally develops at birth or in early life. As such hip dysplasia is screened for, via examination, in all newborns. In hip dysplasia, the acetabulum does not fully cover the femoral head resulting in an increased risk of joint dislocation. If unrecognised it can result in arthritis and movement issues, but again these normal present soon after a child starts walking.



Next question >

Osteoarthritis (OA) of the hip is the second most common presentation of OA after the knee. It accounts for significant morbidity and total hip replacement is now one of the most common operations performed in the developed world.

#### Risk factors

- increasing age
- female gender (twice as common)
- obesity
- developmental dysplasia of the hip

#### **Features**

- chronic history of groin ache following exercise and relieved by rest
- red flag features suggesting an alternative cause include rest pain, night pain and morning stiffness > 2 hours
- the Oxford Hip Score is widely used to assess severity

#### Investigations

- NICE recommends that if the features are typical then a clinical diagnosis can be made
- otherwise plain x-rays are the first-line investigation



#### Management

- oral analgesia
- intra-articular injections: provide short-term benefit
- total hip replacement remains the definitive treatment

### Complications of total hip replacement

- perioperative
  - venous thromboembolism
  - intraoperative fracture
  - nerve injury
  - surgical site infection
- leg length discrepancy
- posterior dislocation
  - may occur during extremes of hip flexion
  - typically presents acutely with a 'clunk', pain and inability to weight bear
  - on examination there is internal rotation and shortening of the affected leg

aseptic loosening (most common reason for revision)
 prosthetic joint infection

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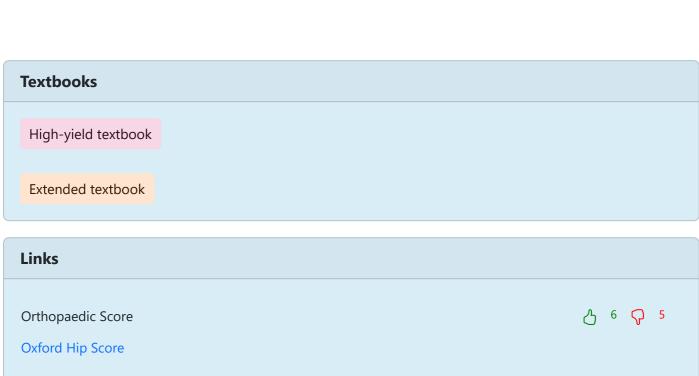
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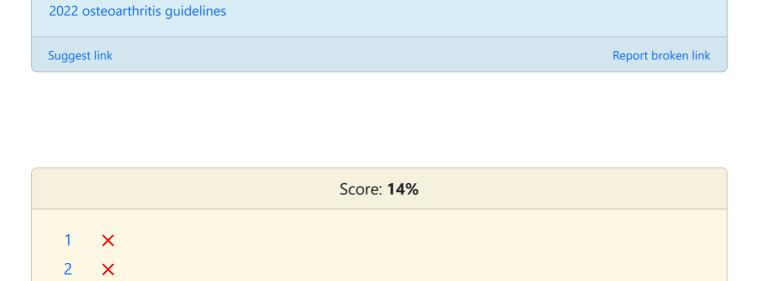
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A 64-year-old man was admitted to the hospital after falling and developing acute pain in his left hip, a fracture was confirmed on CT scan.

His past medical history includes Paget's disease and depression for which he takes venlafaxine. He was treated for acute lymphocytic leukaemia as a child.

The orthopaedic team have asked for some advice as the CT scan has been reported as showing some demineralisation in the femur and pelvis. You recommend a DEXA scan which confirms osteoporosis.

You also request some blood tests which are reported as follows:

Calcium	2.0 mmol/L	(2.1-2.6)
Phosphate	0.9 mmol/L	(0.8-1.4)
Magnesium	0.9 mmol/L	(0.7-1.0)
Thyroid stimulating hormone (TSH)	8.9 mU/L	(0.5-5.5)
Free thyroxine (T4)	3 pmol/L	(9.0 - 18)
Free testosterone	82 ng/dL	(in ages >50 years: 193 - 740)
ALP	202 U/L	(60-306)

What aspect of this man's medical history and blood tests would predispose him to developing osteoporosis?

Acute lymphocytic leukaemia	
Hypothyroidism	
Low testosterone levels	
Paget's disease	
Venlafaxine use	

Submit answer

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 $\Box$ 



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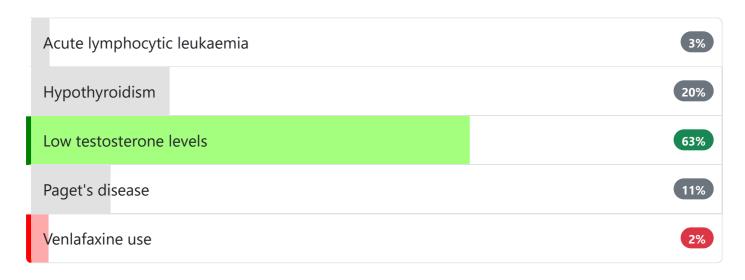
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Free testosterone	82 ng/dL	(in ages >50 years: 193 - 740)
ALP	202 U/L	(60-306)

What aspect of this man's medical history and blood tests would predispose him to developing osteoporosis?



Osteoporosis in a man - check testosterone

**Low testosterone levels**: correct, low testosterone levels are associated with reduced bone mineral density and increased risk of osteoporosis and fractures.

**Acute lymphocytic leukaemia**: multiple myeloma and lymphomas are two haematological malignancies associated with osteoporosis, not acute lymphocytic leukaemia.

**Hypothyroidism**: hyper rather than hypothyroidism is associated with osteoporosis.

**Paget's disease**: this is incorrect, Paget's is not associated with osteoporosis they are entirely separate conditions. Paget's is associated with increased osteoclast activity which leads to increased bone resorption. Osteoblasts respond to this by rapidly increased but abnormal new bone formation.

**Venlafaxine**: SSRIs and anti-epileptics are associated with osteoporosis, but there is not a link with venlafaxine.



Next question >

# Osteoporosis: causes \*

Advancing age and female sex are significant risk factors for osteoporosis. The prevalence of osteoporosis increases from 2% at 50 years to more than 25% at 80 years in women.

There are many other risk factors and secondary causes of osteoporosis. We'll start by looking at the most 'important' ones - these are risk factors that are used by major risk assessment tools such as FRAX:

- history of glucocorticoid use
- · rheumatoid arthritis
- alcohol excess
- history of parental hip fracture
- low body mass index
- current smoking

#### Other risk factors

- sedentary lifestyle
- premature menopause
- Caucasians and Asians
- endocrine disorders: hyperthyroidism, hypogonadism (e.g. Turner's, testosterone deficiency), growth hormone deficiency, hyperparathyroidism, diabetes mellitus
- multiple myeloma, lymphoma

- gastrointestinal disorders: inflammatory bowel disease, malabsorption (e.g. coeliac's), gastrectomy, liver disease
- chronic kidney disease
- osteogenesis imperfecta, homocystinuria

Medications that may worsen osteoporosis (other than glucocorticoids):

- SSRIs
- antiepileptics
- proton pump inhibitors
- glitazones
- long term heparin therapy
- aromatase inhibitors e.g. anastrozole

### Investigations for secondary causes

If a patient is diagnosed with osteoporosis or has a fragility fracture further investigations may be warranted. NOGG recommend testing for the following reasons:

- exclude diseases that mimic osteoporosis (e.g. osteomalacia, myeloma);
- identify the cause of osteoporosis and contributory factors;
- assess the risk of subsequent fractures;
- select the most appropriate form of treatment

The following investigations are recommended by NOGG:

- History and physical examination
- Blood cell count, sedimentation rate or C-reactive protein, serum calcium,

albumin, creatinine, phosphate, alkaline phosphatase and liver transaminases

- Thyroid function tests
- Bone densitometry (DXA)

### Other procedures, if indicated

- Lateral radiographs of lumbar and thoracic spine/DXA-based vertebral imaging
- Protein immunoelectrophoresis and urinary Bence-Jones proteins
- 250HD
- PTH
- Serum testosterone, SHBG, FSH, LH (in men),
- Serum prolactin
- 24 hour urinary cortisol/dexamethasone suppression test
- Endomysial and/or tissue transglutaminase antibodies (coeliac disease)
- Isotope bone scan
- Markers of bone turnover, when available
- Urinary calcium excretion

So from the first list we should order the following bloods as a minimum for all patients:

• full blood count

• urea and electrolytes

• liver function tests

• bone profile

• CRP

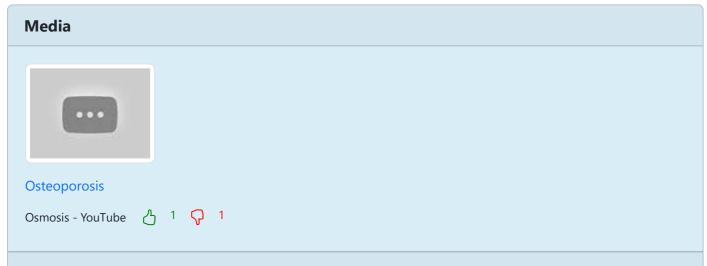
• thyroid function tests





Next question >





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Question 78 of 178





A 78-year-old woman is admitted following a fall. An x-ray of her right wrist showed distal ulnar and radial fractures.

Her past medical history includes diet-controlled diabetes and hypertension.

She wants to know what can be done to prevent further fractures in the future.

What would be the most appropriate investigation and treatment option for her?

Arrange a DEXA scan and commence alendronate depending on the results	
Commence alendronate	
Commence denosumab	
Commence zolendronate	
Start calcium and vitamin D supplementation	

Submit answer

Reference ranges ✓

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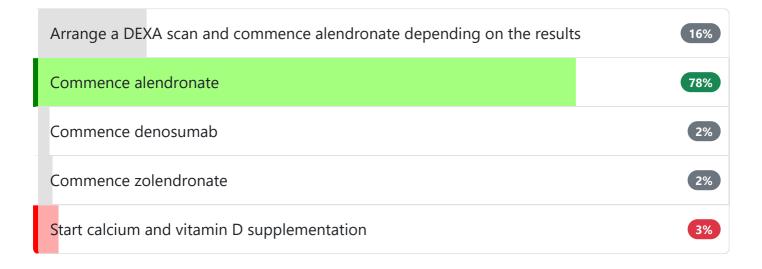


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Her past medical history includes diet-controlled diabetes and hypertension.

She wants to know what can be done to prevent further fractures in the future.

What would be the most appropriate investigation and treatment option for her?



Start alendronate in patients >= 75 years following a fragility fracture, without waiting for a DEXA scan

Important for me Less important

**Commence alendronate** is the correct answer. The patient, in this case, is over 75 years of age and has had a fragility fracture, NICE guidance advises commencing bisphosphonates in this group of patients without waiting for a DEXA scan.

**Commence denosumab** is incorrect. Although this may be an appropriate treatment at some point, it is not yet a first-line treatment for osteoporosis due to cost.

**Arrange a DEXA scan and commence alendronate depending on the results** is incorrect. NICE guidance advises starting bisphosphonates in this group of patients without waiting for a DEXA scan.

**Commence zolendronate** is incorrect. Intravenous bisphosphonates are not generally a first-line option for the treatment of fragility fractures. In some cases, such as following hip fractures, IV zolendronate can be considered a first-line option.

Start calcium and vitamin D supplementation is incorrect. Although this would be

recommended to assist in improving bone health, it is not the most appropriate treatment option for this patient given she has had a fragility fracture already.



Next question >

## Osteoporosis: Assessing patients following a fragility fracture \*



The management of patients following a fragility fracture depends on age.

### Patients >= 75 years of age

Patients who've had a fragility fracture and are >= 75 years of age are presumed to have underlying osteoporosis and should be started on first-line therapy (an oral bisphosphonate), without the need for a DEXA scan.

It should be noted that the 2014 NOGG guidelines have a different threshold, suggesting treatment is started in all women over the age of 50 years who've had a fragility fracture -'although BMD measurement may sometimes be appropriate, particularly in younger postmenopausal women.'

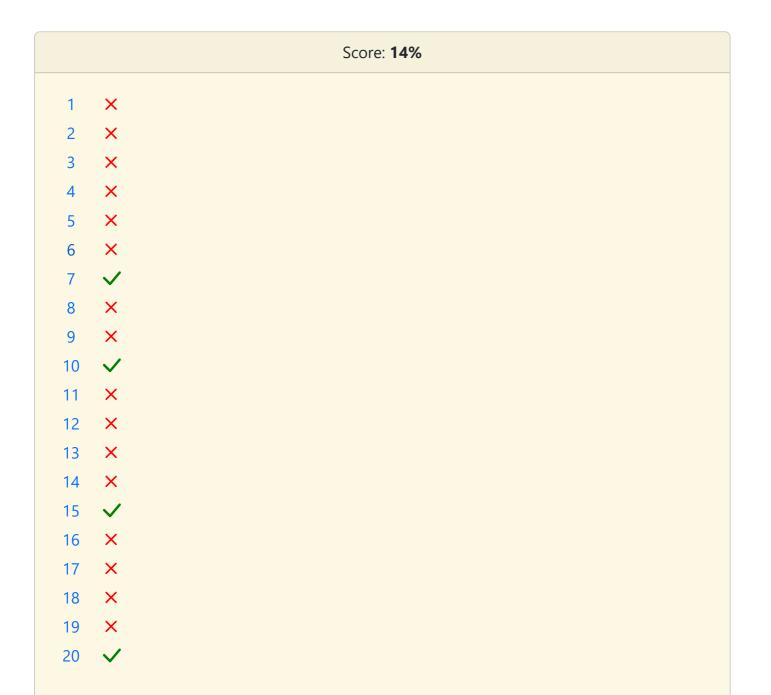
### Patients < 75 years of age

If a patient is under the age of 75 years a DEXA scan should be arranged. These results can then be entered into a FRAX assessment (along with the fact that they've had a fracture) to determine the patients ongoing fracture risk.

For example, a 79-year-old woman falls over on to an outstretched hand and sustains a Colles' fracture (fracture of the distal radius). Given her age she is presumed to have osteoporosis and therefore started on oral alendronate 70mg once weekly. No DEXA scan is arranged.







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An 82-year-old woman presents to the GP with a 2-week history of worsening lower back pain. A lumbar x-ray is organised which reveals a wedge fracture of the L3 vertebral body.

Her past medical history includes hypertension, for which she takes amlodipine, and dietcontrolled type 2 diabetes mellitus.

What is the correct statement regarding this case?

frac	A DEXA scan should be arranged if her QFracture score confirms high risk of fragility ture	
	A DEXA scan should be arranged if she takes regular high-dose corticosteroids	
	A DEXA scan should be arranged regardless of risk factors	
	She should be offered alendronic acid	
	She should be offered referral to a specialist in osteoporosis	

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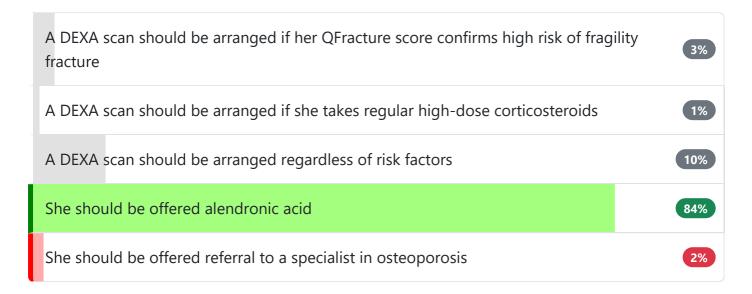
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An 82-year-old woman presents to the GP with a 2-week history of worsening lower back pain. A lumbar x-ray is organised which reveals a wedge fracture of the L3 vertebral body.

Her past medical history includes hypertension, for which she takes amlodipine, and dietcontrolled type 2 diabetes mellitus.

What is the correct statement regarding this case?



Start alendronate in patients >= 75 years following a fragility fracture, without waiting for a DEXA scan

Important for me Less important

The correct answer is **she should be offered alendronic acid**. This patient suffered a fragility fracture over the age of 75. Such patients are generally assumed to have osteoporosis and should be offered bone-sparing treatment. Bisphosphonates such as alendronic acid are recommended first line.

**A DEXA scan should be arranged if her QFracture score confirms the high risk of fragility fracture** is incorrect. Risk scores such as QFracture and FRAX can be used to determine risk in younger patients before organising a DEXA scan. However, over 75, a patient with a fragility fracture should be offered treatment without needing a DEXA.

A DEXA scan should be arranged if she takes regular high-dose corticosteroids is incorrect. Corticosteroids are a risk factor for osteoporosis and this is factored into the risk assessment. However, this patient's risk is sufficiently high by the nature of her age and the presence of a fragility fracture that she should be offered treatment regardless of other risk factors.

A DEXA scan should be arranged regardless of risk factors is incorrect. As the patient is over 75

with a fragility fracture, the risk is sufficiently high to commence treatment here. Delaying treatment for a DEXA scan would not be appropriate.

She should be offered referral to a specialist in osteoporosis is incorrect. This option can be considered in more complex cases, such as young patients at high risk. However, a patient over 75 with a fragility fracture should be offered treatment immediately in primary care - a referral is not necessary.



Next question >

## Osteoporosis: Assessing patients following a fragility fracture \*



The management of patients following a fragility fracture depends on age.

### Patients >= 75 years of age

Patients who've had a fragility fracture and are >= 75 years of age are presumed to have underlying osteoporosis and should be started on first-line therapy (an oral bisphosphonate), without the need for a DEXA scan.

It should be noted that the 2014 NOGG guidelines have a different threshold, suggesting treatment is started in all women over the age of 50 years who've had a fragility fracture -'although BMD measurement may sometimes be appropriate, particularly in younger postmenopausal women.'

### Patients < 75 years of age

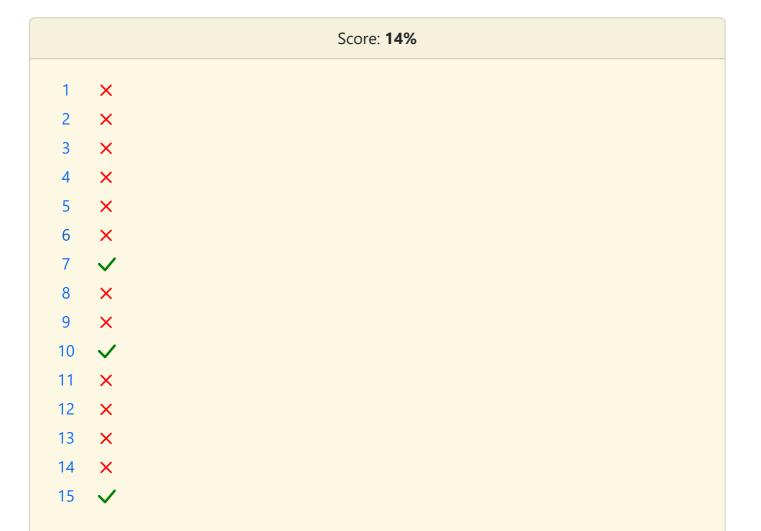
If a patient is under the age of 75 years a DEXA scan should be arranged. These results can then be entered into a FRAX assessment (along with the fact that they've had a fracture) to determine the patients ongoing fracture risk.

For example, a 79-year-old woman falls over on to an outstretched hand and sustains a Colles' fracture (fracture of the distal radius). Given her age she is presumed to have osteoporosis and therefore started on oral alendronate 70mg once weekly. No DEXA scan is arranged.









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Question 80 of 178





A 73-year-old woman attends your clinic with the results of her DEXA scan:

Left neck of femur	-2.8
Right neck of femur	-3.0

Her medical history is notable for previous pulmonary embolism and hyperparathyroidism.

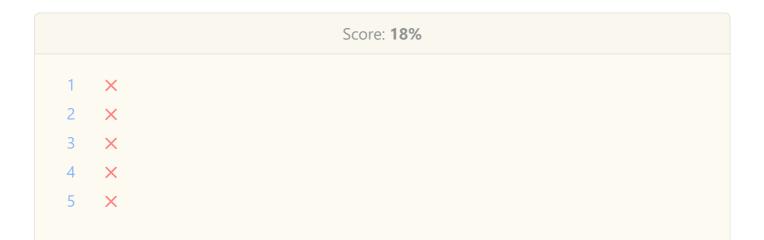
You discuss bone protection, but she developed a severe rash to her bisphosphonates and is not willing to restart any drug of that class.

Which of these describes the mechanism of action of the next most appropriate therapy?

Selective oestrogen receptor modulator (SERM)	
Recombinant parathyroid hormone	
Incorporation into bone in place of calcium	
RANKL inhibitor	
Farnesyl diphosphate synthase (FPPS) inhibitor	

Submit answer

Reference ranges ∨



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Question 80 of 178







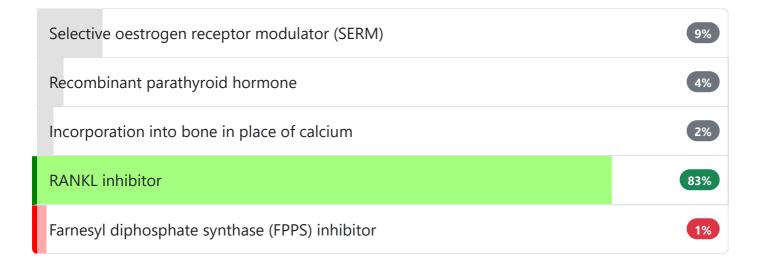
A 73-year-old woman attends your clinic with the results of her DEXA scan:

Left neck of femur	-2.8
Right neck of femur	-3.0

Her medical history is notable for previous pulmonary embolism and hyperparathyroidism.

You discuss bone protection, but she developed a severe rash to her bisphosphonates and is not willing to restart any drug of that class.

Which of these describes the mechanism of action of the next most appropriate therapy?



Denosumab - RANKL inhibitor

Important for me Less important

This woman has critical osteoporosis and is susceptible to a fragility fracture. Many patients have reactions to bisphosphonates and being aware of the contraindications for the numerous other medications is important.

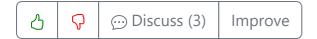
Raloxifene is a SERM and is contraindicated in previous thromboembolic disease. Strontium is also contraindicated in previous venous thromboembolism and works by being incorporated into bone in place of calcium (due to it's atomic similarities).

Teriparatide is a recombinant form of parathyroid hormone and is contraindicated in previous hyperparathyroidism.

FPPS inhibition is the mechanism by which bisphosphonates have their effect.

As such, denosumab is the most appropriate next step. It is a RANKL inhibitor and requires 6

monthly subcutaneous injections. It is recommended by NICE for patients who are unable to tolerate or are precluded from the other options.



Next question >

### Denosumab \*

Denosumab is a relatively new treatment for osteoporosis. It is a human monoclonal antibody that prevents the development of osteoclasts by inhibiting RANKL. Remember that osteoblasts build bone, osteoclasts eat bone. It is given as a subcutaneous injection, at a dose of 60mg, every 6 months.

A larger dose of denosumab (120mg) may also be given every 4 weeks for the prevention of skeletal-related events (i.e. pathological fractures) in adults with bone metastases from solid tumours. For example, you may have noticed some of your breast cancer patients have been prescribed denosumab.

### Where does it fit in the management of osteoporosis?

Oral bisphosphonates are still given first-line, with oral alendronate being the first-line treatment. If alendronate is not tolerated then NICE recommend using an alternative bisphosphonate - either risedronate or etidronate. Following this the advice becomes more complicated with the next-line medications only being started if certain T score and other risk factor criteria being met. Raloxifene and strontium ranelate were recommended as next-line drugs in the NICE criteria but following recent safety concerns regarding strontium ranelate it is likely there will be an increasing role for denosumab.

NICE published a technology appraisal looking at the role of denosumab in 2010. A link is provided.

### What are the known side-effects of denosumab?

Denosumab is generally well tolerated. Dyspnoea and diarrhoea are generally considered the two most common side effects, occuring in around 1 in 10 patients. Other less common side effects include hypocalcaemia and upper respiratory tract infections.

### What does the Drug Safety Update add?

Cases of atypical femoral fractures have been noted in patients taking denosumab. Doctors are advised to look out for patients complaining of unusual thigh, hip or groin pain.

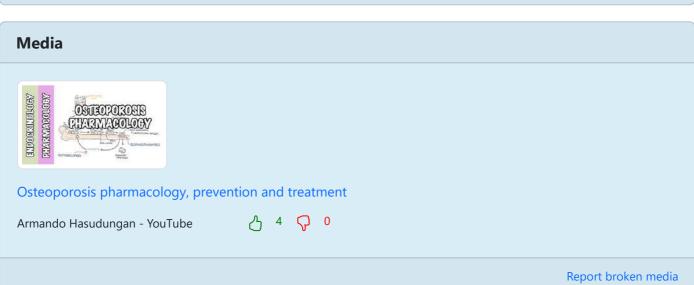


Next question >









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#### Ouestion 81 of 178

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A 72-year-old woman presents to the emergency department because she is struggling to cope at home. She has noticed that she has been struggling to get out of bed over the last month and not been feeling herself. She denies any fevers or specific systemic symptoms other than feeling weak and tired and her arms have been feeling heavy in the morning. Both her shoulders have been aching, and her appetite has been reduced. She has a past medical history of stroke, from which she made a complete recovery, atrial fibrillations, heart attack with coronary stenting and hypertension. Her observations are normal apart from a temperature of 37.7 degrees. Her joints are not tender and not swollen. She has normal power in her shoulders. Her visual acuity is normal.

#### Blood tests:

Hb	122 g/l	
Platelets	366 * 10 <sup>9</sup> /l	
WBC	8.9 * 10 <sup>9</sup> /l	
Na <sup>+</sup>	137 mmol/l	
K <sup>+</sup>	4.1 mmol/l	
Urea	6.2 mmol/l	
Creatinine	122 µmol/l	

What is the most likely diagnosis?

Polymyalgia rheumatica	
Rheumatoid arthritis	
Dermatomyositis	
Osteoarthritis	
Adhesive capsulitis	

Submit answer

Reference ranges ∨

#### Score: **18%**

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A 72-year-old woman presents to the emergency department because she is struggling to cope at home. She has noticed that she has been struggling to get out of bed over the last month and not been feeling herself. She denies any fevers or specific systemic symptoms other than feeling weak and tired and her arms have been feeling heavy in the morning. Both her shoulders have been aching, and her appetite has been reduced. She has a past medical history of stroke, from which she made a complete recovery, atrial fibrillations, heart attack with coronary stenting and hypertension. Her observations are normal apart from a temperature of 37.7 degrees. Her joints are not tender and not swollen. She has normal power in her shoulders. Her visual acuity is normal.

#### Blood tests:

Hb	122 g/l	
Platelets	366 * 10 <sup>9</sup> /l	
WBC	8.9 * 10 <sup>9</sup> /I	
Na <sup>+</sup>	137 mmol/l	
K <sup>+</sup>	4.1 mmol/l	
Urea	6.2 mmol/l	
Creatinine	122 µmol/l	

What is the most likely diagnosis?

Polymyalgia rheumatica	91%
Rheumatoid arthritis	1%
Dermatomyositis	3%
Osteoarthritis	3%
Adhesive capsulitis	2%

Polymyalgia rheumatic is characterised by abrupt onset of bilateral early morning stiffness in the over 60s

Important for me Less important

The most likely diagnosis is polymyalgia rheumatica due to the sudden onset of bilateral proximal difficulty in moving arms in a patient over 50 years old without weakness. This is commonly, but not always, described as a stiffness. These are some of the key features of polymyalgia rheumatica

which help diagnosis, and the presence of loss of appetite, low-grade fever and morning symptoms are supportive. Dermatomyositis is a differential diagnosis that can be hard to exclude but the absence of rash, detectable weakness and muscle tenderness makes this unlikely. Rheumatoid arthritis would be unusual at this age without symmetrical polyarthritis but the acute onset makes it even less likely. Osteoarthritis normally affects hips and knees to a greater extent and would have a gradual onset without fever. Adhesive capsulitis is possible as a bilateral diagnosis but would need a reduced range of motion in both passive and active ranges and would not be as systemic as in this patient.



Next question >

# Polymyalgia rheumatica 🖈

Polymyalgia rheumatica (PMR) is a relatively common condition seen in older people characterised by muscle stiffness and raised inflammatory markers. Whilst it appears to be closely related to temporal arteritis the underlying cause is not fully understood and it does not appear to be a vasculitic process.

#### **Features**

- typically patient > 60 years old
- usually rapid onset (e.g. < 1 month)</li>
- aching, morning stiffness in proximal limb muscles
  - weakness is not considered a symptom of polymyalgia rheumatica
- also mild polyarthralgia, lethargy, depression, low-grade fever, anorexia, night sweats

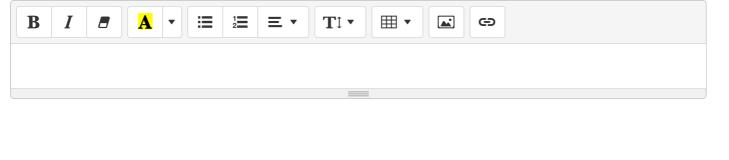
#### Investigations

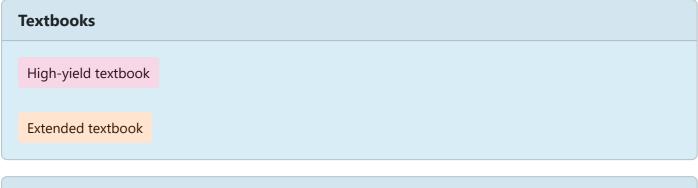
- raised inflammatory markers e.g. ESR > 40 mm/hr
- note creatine kinase and EMG normal

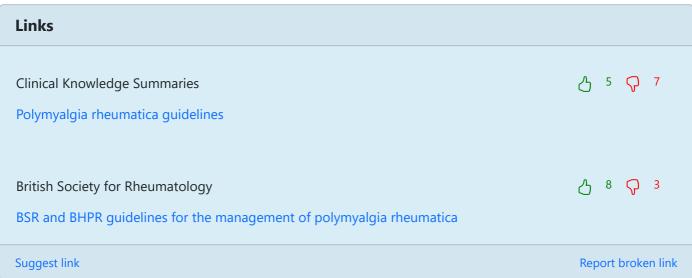
#### **Treatment**

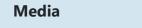
- prednisolone e.g. 15mg/od
  - patients typically respond dramatically to steroids, failure to do so should prompt consideration of an alternative diagnosis













Polymyalgia rheumatica

Zero To Finals - YouTube





Polymyalgia rheumatica

Osmosis - YouTube









#### Polymyalgia rheumatica

Townsend Teaching - YouTube  $\bigcirc$  0  $\bigcirc$  0







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Question 82 of 178





A 68-year-old female diagnosed with rheumatoid arthritis four years ago presents gradually increasing tenderness in the small joints of both hands over the past 5 months. She continues to work as a legal secretary, involving significant amounts of time at a computer. She is currently on maximum doses of methotrexate and sulphasalazine on diagnosis and maintained on the same doses since. Her DAS score today is 5.8, it was 4.7 when you saw her in clinic last 1 month ago. What is the next management step?

Continue methotrexate and sulphasalazine. Short-course oral prednisolone	
Stop current DMARDs. Start etanercept	
Stop current DMARDs. Start infliximab	
Admit for pulsed intravenous methylprednisolone	
Prescribe regular long-term celecoxib in addition to methotrexate and sulphasalazine	

Submit answer

Reference ranges ∨

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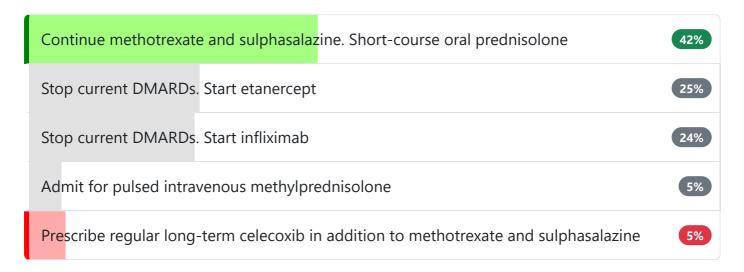
Question 82 of 178



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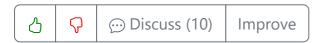
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A 68-year-old female diagnosed with rheumatoid arthritis four years ago presents gradually increasing tenderness in the small joints of both hands over the past 5 months. She continues to work as a legal secretary, involving significant amounts of time at a computer. She is currently on maximum doses of methotrexate and sulphasalazine on diagnosis and maintained on the same doses since. Her DAS score today is 5.8, it was 4.7 when you saw her in clinic last 1 month ago. What is the next management step?



Current NICE guidelines recommend the starting of biologic therapy when the patient has been on at least two DMARDs, including methotrexate, reporting two DAS 28 scores of greater than 5.1 at least one month apart<sup>1</sup>. A short course of oral prednisolone may be appropriate for flares for symptomatic control. However, this is not an option if the patient does not wish to take any steroids. Intravenous pulsed steroids or long-term treatments are not appropriate. Regular COX-2 inhibitors are not recommended by NICE guidelines. NSAIDs are appropriate for short-term symptomatic control but only at lowest doses for as short a period as possible.

1. NICE Clinical Guideline 79. The management of rheumatoid arthritis in adults. Jan 2009



Next question >

# Rheumatoid arthritis: management \*

The management of rheumatoid arthritis (RA) has been revolutionised by the introduction of disease-modifying therapies in the past decade.

Patients with evidence of joint inflammation should start a combination of disease-modifying

drugs (DMARD) as soon as possible. Other important treatment options include analgesia, physiotherapy and surgery.

#### Initial therapy

- NICE recommend DMARD monotherapy +/- a short-course of bridging prednisolone. In the past dual DMARD therapy was advocated as the initial step
- choices for initial DMARD monotherapy:
  - **methotrexate** is the most widely used DMARD. Monitoring of FBC & LFTs is essential due to the risk of myelosuppression and liver cirrhosis. Other important side-effects include pneumonitis
  - o sulfasalazine
  - o leflunomide
  - hydroxychloroquine: should only be considered for initial therapy if mild or palindromic disease

#### Monitoring response to treatment

 NICE recommends using a combination of CRP and disease activity (using a composite score such as DAS28) to assess response to treatment

#### Flares

• flares of RA are often managed with corticosteroids - oral or intramuscular

#### TNF-inhibitors

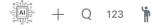
- the current indication for a TNF-inhibitor is an inadequate response to at least two DMARDs including methotrexate
- etanercept: recombinant human protein, acts as a decoy receptor for TNF- $\alpha$ , subcutaneous administration, can cause demyelination, risks include reactivation of tuberculosis
- infliximab: monoclonal antibody, binds to TNF- $\alpha$  and prevents it from binding with TNF receptors, intravenous administration, risks include reactivation of tuberculosis
- adalimumab: monoclonal antibody, subcutaneous administration

#### Rituximab

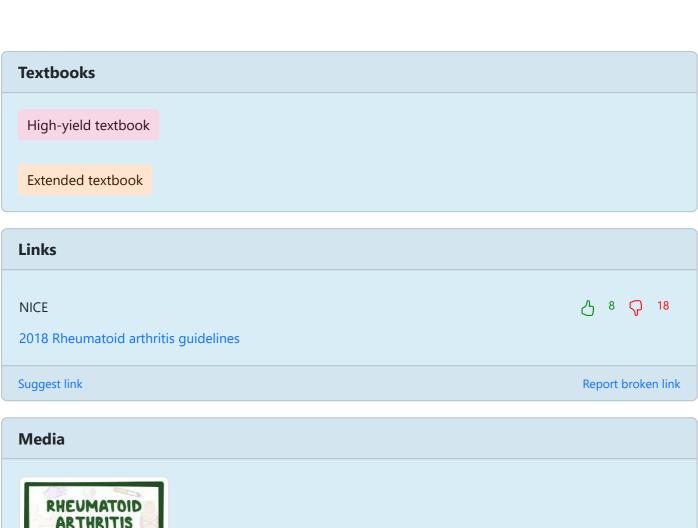
- anti-CD20 monoclonal antibody, results in B-cell depletion
- two 1g intravenous infusions are given two weeks apart
- infusion reactions are common

#### Abatacept

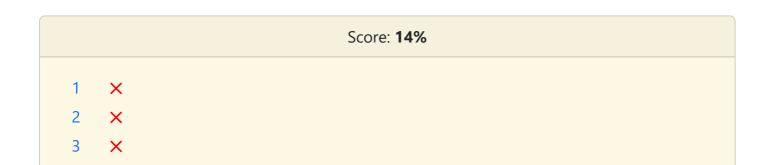
- fusion protein that modulates a key signal required for activation of T lymphocytes
- leads to decreased T-cell proliferation and cytokine production
- given as an infusion
- not currently recommend by NICE











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Question 83 of 178





A 54-year-old woman presents to her general practitioner with three days of right shoulder pain and stiffness. She has a past medical history of type 2 diabetes and takes metformin and gliclazide. She does not smoke or drink alcohol. She works as a screenwriter.

On examination, she has globally restricted active and passive movements of the right shoulder. External rotation is most affected. There is no crepitus.

Given the likely diagnosis, what is the best method of confirming the diagnosis?

Arthroscopy	
Clinical examination	
MRI	
Ultrasound	
X-ray	

Submit answer

Reference ranges ∨

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Question 83 of 178



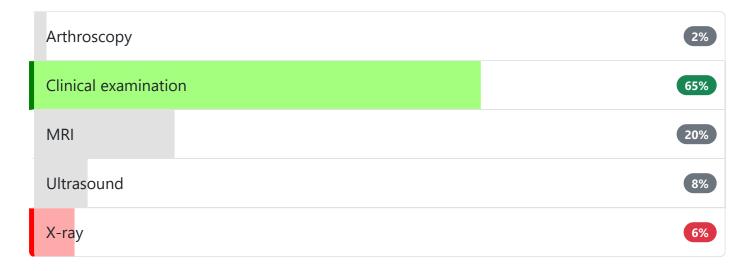
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A 54-year-old woman presents to her general practitioner with three days of right shoulder pain and stiffness. She has a past medical history of type 2 diabetes and takes metformin and gliclazide. She does not smoke or drink alcohol. She works as a screenwriter.

On examination, she has globally restricted active and passive movements of the right shoulder. External rotation is most affected. There is no crepitus.

Given the likely diagnosis, what is the best method of confirming the diagnosis?



Adhesive capsulitis is a clinical diagnosis and does not need imaging or arthroscopy to confirm the diagnosis

Important for me Less important

Clinical examination is the correct answer. The patient presents with three days of shoulder stiffness and pain. The clue to the diagnosis is that shoulder movement is limited both on active and passive movement, suggesting adhesive capsulitis. The typical differential diagnosis for shoulder pain and stiffness includes rotator cuff disorders, osteoarthritis and adhesive capsulitis. Rotator cuff disorders principally limit active movement. While osteoarthritis may cause pain on active and passive movement, typically the joint can still be moved through its range of motion on passive movement, unlike in adhesive capsulitis where the stiffness cannot be overcome. Adhesive capsulitis is more common in females and those with type 2 diabetes. It is a clinical diagnosis and does not require imaging or arthroscopy.

**Arthroscopy** is the gold standard investigation to diagnose many shoulder disorders, allowing the surgeon direct visualization of anatomic structures. However, it is an invasive procedure and therefore is only indicated if conventional imaging is insufficient to make a diagnosis or where intervention is expected.

MRI can be used to diagnose a wide variety of shoulder disorders, including adhesive capsulitis.

However, its use is limited by cost. Given that adhesive capsulitis can be diagnosed clinically, it is superfluous in this case.

**Ultrasound** is typically used to diagnose pathology associated with rotator cuff disorders e.g. supraspinatus tendinopathy or tear. Examination findings in rotator cuff disorders typically affect active movements rather than passive movements and so a cuff disorder is unlikely in this case.

**X-ray** can be used to diagnose osteoarthritis and fractures. The examination findings, in this case, suggest adhesive capsulitis rather than osteoarthritis. Osteoarthritis would be a more indolent history than a three-day onset of pain and stiffness. Additionally, the degree of restriction, in this case, favours adhesive capsulitis.



Next question >

# Adhesive capsulitis \*

Adhesive capsulitis (frozen shoulder) is a common cause of shoulder pain. It is most common in middle-aged females. The aetiology of frozen shoulder is not fully understood.

#### Associations

diabetes mellitus: up to 20% of diabetics may have an episode of frozen shoulder

Features typically develop over days

- external rotation is affected more than internal rotation or abduction
- both active and passive movement is affected
- patients typically have a painful freezing phase, an adhesive phase and a recovery phase
- bilateral in up to 20% of patients
- the episode typically lasts between 6 months and 2 years

The diagnosis is usually clinical although imaging may be required for atypical or persistent symptoms.

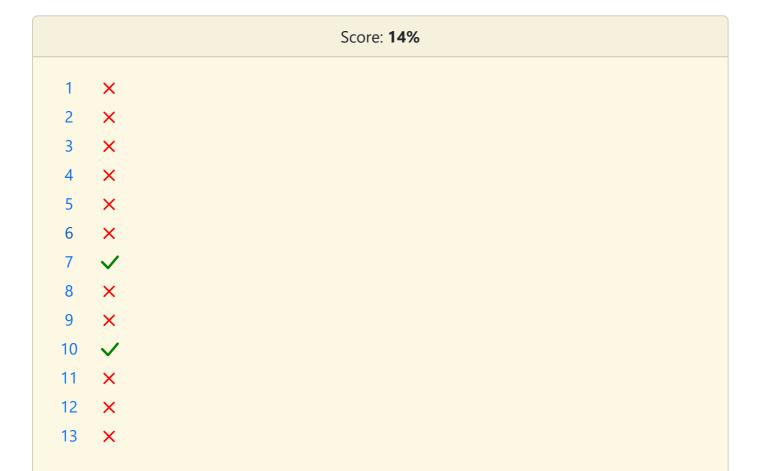
#### Management

- no single intervention has been shown to improve outcome in the long-term
- treatment options include NSAIDs, physiotherapy, oral corticosteroids and intra-articular corticosteroids









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6

A 56-year-old man presents with a scaly rash affecting the back of his hands. The erythematous rash is present on the extensor aspects of his fingers particularly located over the MCP and PIP joints. In addition, he has noticed a violaceous swelling of his left upper eyelid. These have troubled him for the last two weeks and he has trialled an emollient cream without any benefit. His two brothers both have psoriasis and apart from childhood eczema he has been fit and well since.

In addition to these rashes, he also has a palpable mass in the left iliac fossa that is non tender and a nodular liver edge can be felt in the right upper quadrant. His conjunctiva are pale. He reports that his bowels can vary between diarrhoea and occasional constipation. He had started taking mebeverine but did not find any benefit. There are no other rashes present.

What is the likely nature of this rash?

Hb	94 g/l	Na <sup>+</sup>	138 mmol/l
Platelets	400 * 10 <sup>9</sup> /l	K <sup>+</sup>	5.0 mmol/l
WBC	3.9 * 10 <sup>9</sup> /l	Urea	6.0 mmol/l
Neuts	3.1* 10 <sup>9</sup> /I	Creatinine	80 µmol/l
MCV	69 fL (range 80-100)		
Ferritin	2 ng/ml (range 20-230)		
Creatine kinase	340 U/L (range 40-320)		

Paraneoplastic dermatomyositis	
Atopic eczema	×
Chronic plaque psoriasis	×
Erythema gyratum repens	×
Idiopathic dermatomyositis	

Submit answer

Reference ranges ∨

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Question 84 of 178



 $\Box$ 



A 56-year-old man presents with a scaly rash affecting the back of his hands. The erythematous rash is present on the extensor aspects of his fingers particularly located over the MCP and PIP joints. In addition, he has noticed a violaceous swelling of his left upper eyelid. These have troubled him for the last two weeks and he has trialled an emollient cream without any benefit. His two brothers both have psoriasis and apart from childhood eczema he has been fit and well since.

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MCV	69 fL (range 80-100)		
Ferritin	2 ng/ml (range 20-230)		
Creatine kinase	340 U/L (range 40-320)		

Paraneoplastic dermatomyositis	79%
Atopic eczema	1%
Chronic plaque psoriasis	3%
Erythema gyratum repens	6%
Idiopathic dermatomyositis	11%

Dermatomyositis is commonly a paraneoplastic phenomenon

Important for me Less important

The description gives Gottron's papules on the hand and a heliotrope rash on the face. This is pathognomonic for dermatomyositis which can be idiopathic but is commonly secondary to

malignancy. The abdominal mass could be a rectal tumour and the nodules in the liver may be metastases. Erythema gyratum repens is another paraneoplastic rash that can occur but is very distinctive and covers large areas.



Next question >

# Dermatomyositis \*

#### Overview

- an inflammatory disorder causing symmetrical, proximal muscle weakness and characteristic skin lesions
- may be idiopathic or associated with connective tissue disorders or underlying malignancy (typically ovarian, breast and lung cancer, found in 20-25% more if patient older). Screening for an underlying malignancy is usually performed following a diagnosis of dermatomyositis
- polymyositis is a variant of the disease where skin manifestations are not prominent

#### Skin features

- photosensitive
- macular rash over back and shoulder
- heliotrope rash in the periorbital region
- Gottron's papules roughened red papules over extensor surfaces of fingers
- 'mechanic's hands': extremely dry and scaly hands with linear 'cracks' on the palmar and lateral aspects of the fingers
- nail fold capillary dilatation

#### Other features

- proximal muscle weakness +/- tenderness
- Raynaud's
- respiratory muscle weakness
- interstitial lung disease: e.g. Fibrosing alveolitis or organising pneumonia
- dysphagia, dysphonia

#### Investigations

- the majority of patients (around 80%) are ANA positive
- around 30% of patients have antibodies to aminoacyl-tRNA synthetases (anti-synthetase antibodies), including:
  - o antibodies against histidine-tRNA ligase (also called Jo-1)
  - antibodies to signal recognition particle (SRP)
  - o anti-Mi-2 antibodies



Next question >







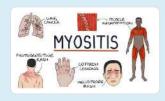
#### Media

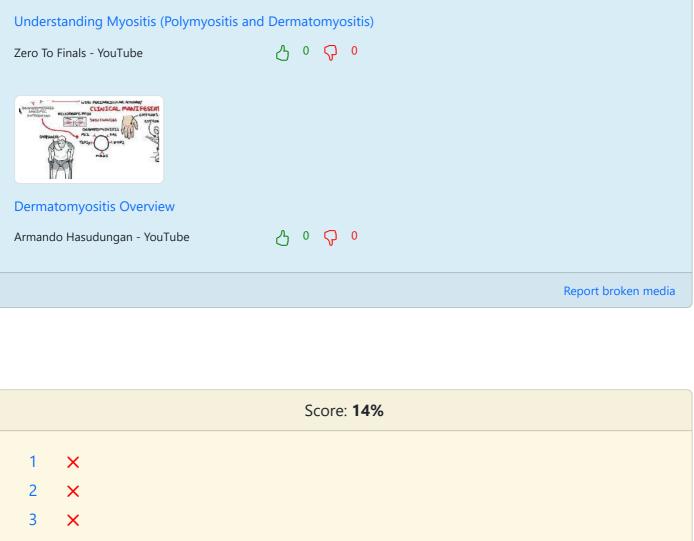


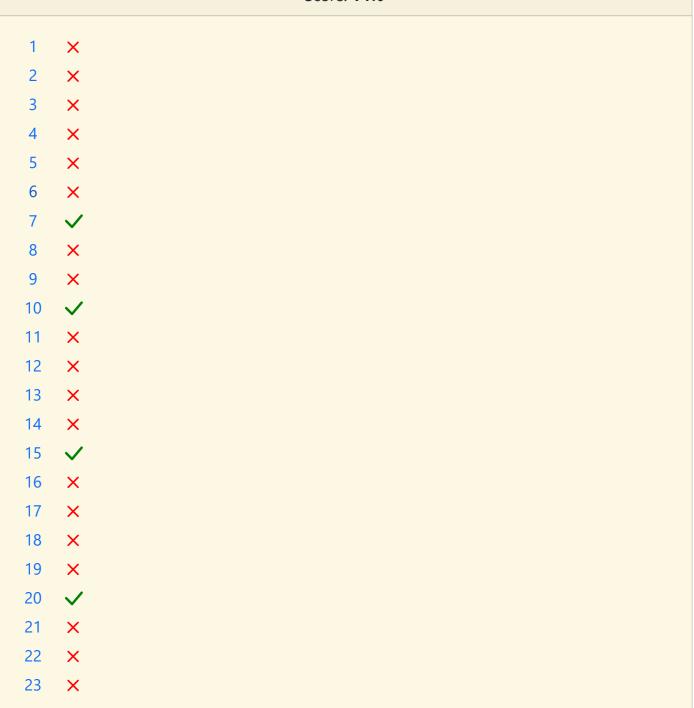
Polymyositis and Dermatomyositis in 3 Minutes

Townsend Teaching - YouTube









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A 24-year-old lady attends her GP with a cough for the past 4 days. She is normally well but has been suffering knee pains and has started ibuprofen and physio for her knee. During her recovery, she started exercising at the gym and noted that she felt breathless and her chest was tight. This has been followed by a cough which is worse at night and is non productive. She sleeps with two pillows and has no ankle swelling. She is normally fit and well and only has been seen as a child for breathing problems in which she took inhalers but these have long since resolved. She admits to smoking 20 cigarettes a day for the past ten years. She takes no regular medication at present and denies any foreign travel or ill contacts.

On examination, she has a dry cough and is able to speak in full sentences. She has a clear chest with mild wheeze heard in the left lower base. Her peak flow is 220 and she saturates at 98% in air with a respiratory rate of 21 breaths per minute.

Na <sup>+</sup>	139 mmol/l
K <sup>+</sup>	4.2 mmol/l
Urea	5.1 mmol/l
Creatinine	68 µmol/l

Hb	110 g/l	
Platelets	390 * 10 <sup>9</sup> /I	
WBC	10.0 * 10 <sup>9</sup> /l	

Chest x-ray clear lung fields with good chest expansion, no active lung lesion

What is the likely diagnosis?

Bronchiectasis	
Asthma	
COPD	
Anxiety	
Occult pneumothorax	

Submit answer

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 $\Box$ 



A 24-year-old lady attends her GP with a cough for the past 4 days. She is normally well but has been suffering knee pains and has started ibuprofen and physio for her knee. During her recovery, she started exercising at the gym and noted that she felt breathless and her chest was tight. This has been followed by a cough which is worse at night and is non productive. She sleeps with two pillows and has no ankle swelling. She is normally fit and well and only has been seen as a child for breathing problems in which she took inhalers but these have long since resolved. She admits to smoking 20 cigarettes a day for the past ten years. She takes no regular medication at present and denies any foreign travel or ill contacts.

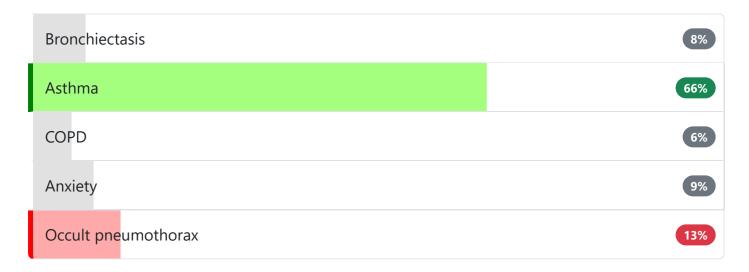
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K <sup>+</sup>	4.2 mmol/l	
Urea	5.1 mmol/l	
Creatinine	68 µmol/l	

Hb	110 g/l	
Platelets	390 * 10 <sup>9</sup> /I	
WBC	10.0 * 10 <sup>9</sup> /l	

Chest x-ray clear lung fields with good chest expansion, no active lung lesion

What is the likely diagnosis?



This lady has developed SOB and a cough which are new for her. Although she smokes, it is unlikely for her to have COPD unless she had alpha-1-antitrypsin deficiency which is rare. She could have bronchiectasis given that it is caused by viral and other respiratory illnesses in children but this presents with a productive cough and infection. An occult pneumothorax is unlikely and does not explain tightness or the nocturnal cough. Her peak flow is clearly reduced and this suggests it is not just anxiety. Given that she has previously taken inhalers as a child and now has developed a tight chest and reduced peak flow after starting and NSAID, it is likely this is asthma. Note that asthma does not always have audible wheeze and most investigations are normal.



Next question >

# Non-steroidal anti-inflammatory drugs (NSAIDs)

NSAIDs work by inhibiting the activity of cyclooxygenase enzymes (COX-1 and/or COX-2) thus reducing the production of key mediators involved in inflammation such as prostaglandins. They also have an antipyretic action as prostaglandin E2 is involved in the thermoregulation centre in the hypothalamus.

Examples of NSAIDs include

- ibuprofen
- diclofenac
- naproxen
- aspirin

Important and common side-effects

- peptic ulceration
- exacerbation of asthma

### COX-2 selective NSAIDs

COX-2 selective NSAIDs were developed to reduce the incidence of side-effects seen with traditional NSAIDs, in particular, peptic ulceration. Examples include celecoxib and etoricoxib. Coxibs are not widely used due to ongoing concerns about cardiovascular safety. This led to the withdrawal of rofecoxib ('Vioxx') in 2004.

Cardiovascular adverse effects of celecoxib:

• increased risk of thrombotic effects → myocardial infarction and stroke

- fluid retention → exacerbation of existing heart failure
- worsening of hypertension



Next question >



# **Textbooks**

High-yield textbook

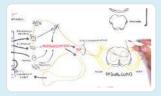
Extended textbook

# Media



NSAIDs & prostaglandin analogs

Speed Pharmacology - YouTube 💍 1 🖓 0



Nonsteroidal Anti-Inflammatory Drugs (NSAIDs)

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A 75-year-old gentleman presents to the emergency department complaining about left eye visual disturbance with peripheral vision loss noted. He tells you that over the last 2 weeks he has had constant headache, worse on the left side of his skull. It is worse when he applies pressure to it and has noted that he cannot lie on that side.

Examination is consistent with peripheral loss of vision (temporal lower quadrant) of his left eye. In addition he is quite tender over the left temporal region, with pain on his shoulders as well. He is otherwise neurologically intact but it appears that he has mild difficulty in rising from sitting.

His bloods from today include:

Hb	131 g/l	Na <sup>+</sup>	133 mmol/l
Platelets	378 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.4 mmol/l
WBC	9.2 * 10 <sup>9</sup> /l	Urea	8.9 mmol/l
Neuts	5.6 * 10 <sup>9</sup> /l	Creatinine	98 µmol/l
Lymphs	3.1 * 10 <sup>9</sup> /l	CRP	133 mg/l
Eosin	0.4 * 10 <sup>9</sup> /l	ESR	79 mm/h

He is obviously concerned regarding his symptoms and is wondering what needs to be done next:

rhe	Discharge home with oral steroids (prednisolone 60 mg OD) and arrange outpatient umatology review	
	Arrange for review by ophthalmology on next opportunity that arises	
	Organise urgent CT head	
	Admit to hospital urgently for IV methylprednisolone infusion	
	Prescribe aspirin 300 mg OD STAT	

Submit answer

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 $\Box$ 



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Eosin	0.4 * 10 <sup>9</sup> /l	ESR	79 mm/h

He is obviously concerned regarding his symptoms and is wondering what needs to be done next:

Discharge home with oral steroids (prednisolone 60 mg OD) and arrange outpatient rheumatology review	7%
Arrange for review by ophthalmology on next opportunity that arises	2%
Organise urgent CT head	1%
Admit to hospital urgently for IV methylprednisolone infusion	90%
Prescribe aspirin 300 mg OD STAT	1%

Urgent admission and administration of corticosteroids is important in giant cell arteritis associated with visual loss

Important for me Less important

Given the history this patient is suffering from temporal arteritis (giant cell arteritis) with visual loss. Such cases represent major emergencies that require urgent admission to hospital for IV

administration of steroids. Any delay to its management can lead to permanent visual loss. It is understandable that one that one may be concerned regarding a transient ischaemic attack or indeed a cerebrovascular accident and be tempted to organise an urgent CT of the head, however the history, examination findings and blood investigations point more towards the direction of giant cell arteritis.



Next question >

# Temporal arteritis \*

Temporal arteritis (also known as giant cell arteritis: GCA) is a vasculitis of unknown cause that affects medium and large-sized vessels arteries. It occurs in those over 50 years old, with a peak incidence in patients who are in their 70s.

It requires early recognition and treatment to minimize the risk of complications such as permanent loss of vision. Hence, when temporal arteritis is suspected, treatment must be started promptly with high-dose prednisolone as well as urgent referral for assessment by a specialist.

There is an overlap between temporal arteritis and polymyalgia rheumatica (PMR) - around 50% of patients will have features of PMR.

#### **Features**

- typically patient > 60 years old
- usually rapid onset (e.g. < 1 month)
- headache (found in 85%)
- jaw claudication (65%)
- vision testing is a key investigation in all patients
  - anterior ischemic optic neuropathy accounts for the majority of ocular complications. It results from occlusion of the posterior ciliary artery (a branch of the ophthalmic artery) → ischaemia of the optic nerve head. Fundoscopy typically shows a swollen pale disc and blurred margins
  - o may result in temporary visual loss amaurosis fugax
  - permanent visual loss is the most feared complication of temporal arteritis and may develop suddenly
  - diplopia may also result from the involvement of any part of the oculomotor system (e.g. cranial nerves)
- tender, palpable temporal artery
- around 50% have features of PMR: aching, morning stiffness in proximal limb muscles (not weakness)
- also lethargy, depression, low-grade fever, anorexia, night sweats

## Investigations

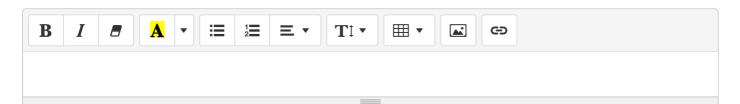
- raised inflammatory markers
  - ESR > 50 mm/hr (note ESR < 30 in 10% of patients)</li>
  - CRP may also be elevated
- temporal artery biopsy
  - o skip lesions may be present
- note creatine kinase and EMG normal

#### Treatment

- urgent high-dose glucocorticoids should be given as soon as the diagnosis is suspected and before the temporal artery biopsy
  - o if there is no visual loss then high-dose prednisolone is used
  - o if there is evolving visual loss IV methylprednisolone is usually given prior to starting high-dose prednisolone
  - there should be a dramatic response, if not the diagnosis should be reconsidered
- urgent ophthalmology review
  - o patients with visual symptoms should be seen the same-day by an ophthalmologist
  - o visual damage is often irreversible
- other treatments
  - bone protection with bisphosphonates is required as long, tapering course of steroids is required
  - low-dose aspirin is sometimes given to patients as well, although the evidence base supporting this is weak

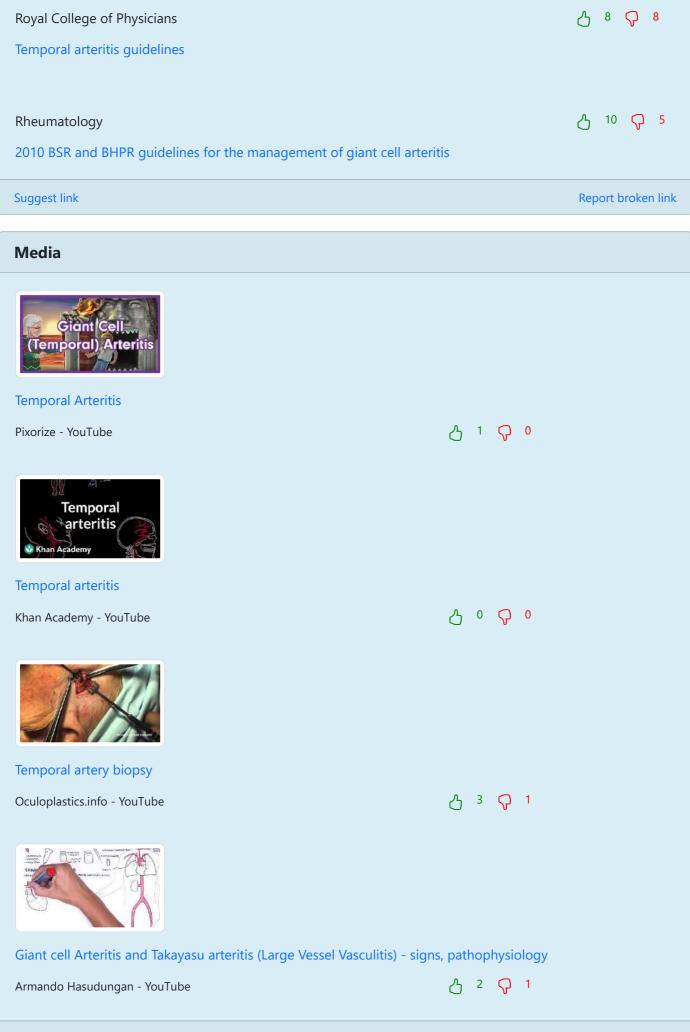


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# Textbooks High-yield textbook Extended textbook

#### Links



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A 59-year-old lady comes to the blood pressure clinic with accelerated hypertension which isn't responding to lifestyle modifications.

The patient denies any headaches or blurred vision. However, she admits to a chronic cough and frequently passes pale, loose stools. She frequently gets cold hands and tells you they go red, white and then blue in the winter.

On examination, she is a thin lady with a blood pressure of 190/100 mmHg and a heart rate of 68 beats per minute. Although her nails are a normal colour she has tight, shiny skin over her hands. An ECG shows sinus rhythm.

Hb	106 g/l	
Platelets	451 * 10 <sup>9</sup> /l	
WBC	8.9 * 10 <sup>9</sup> /I	
Na <sup>+</sup>	136 mmol/l	
K <sup>+</sup>	4.9 mmol/l	
Urea	7.1 mmol/l	
Creatinine	174 µmol/l	

Which of the following is the most appropriate initial therapy?

Bisoprolol	
Candesartan	
Captopril	
Indapamide	
Amlodipine	

Submit answer

Reference ranges ∨

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The patient denies any headaches or blurred vision. However, she admits to a chronic cough and frequently passes pale, loose stools. She frequently gets cold hands and tells you they go red, white and then blue in the winter.

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K <sup>+</sup>	4.9 mmol/l	
Urea	7.1 mmol/l	
Creatinine	174 µmol/l	

Which of the following is the most appropriate initial therapy?

Bisoprolol	1%
Candesartan	5%
Captopril	74%
Indapamide	1%
Amlodipine	18%

The patient describes Raynaud's phenomenon and mentions bowel and possibly lung involvement along with tight skin. This could point towards scleroderma. In this case, then, the raised creatinine and hypertension are suggestive of a scleroderma renal crisis. The first line treatment, in this case, is an ACE inhibitor.

Amlodipine would be the antihypertensive of choice if this were essential hypertension.

Next question >

# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - patients with renal disease should be started on an ACE inhibitor
- poor prognosis

Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







#### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis

- o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis



Next question >











#### Systemic Sclerosis and Scleroderma

Zero to Finals - YouTube

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A 41-year-old woman presents with tightening of fingers, mild difficulty swallowing, and mild shortness of breath on exertion. She takes pantoprazole for reflux. On examination there is tightening of skin in her fingers, however the rest of the skin is normal. Her joints are not inflamed. The rest of her examination was normal (including chest examination). Her chest X-ray is also normal. There is mild decrease in DLCO on lung function tests. Which of the following antibodies are indicative of the underlying diagnosis?

Anti-Scl-70 antibody	
Anti-dsDNA antibody	
Rh factor antibody	
Anti-centromere antibody	
Anti-Jo-1 antibody	

Submit answer

Reference ranges  $\vee$ 

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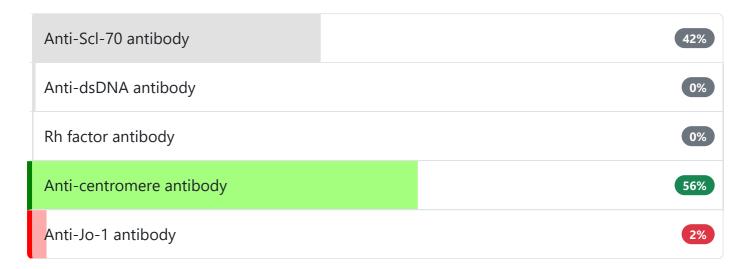
Question 88 of 178



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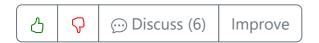


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The underlying diagnosis is limited scleroderma. Limited scleroderma has tightened hard skin below the elbows. Dysphagia and some lung fibrosis occurs in both limited and diffuse scleroderma. Limited scleroderma is associated with anti-centromere antibody with Anti-scl-70 antibody associated with diffuse scleroderma. Diffuse scleroderma is characterised by lesions proximal to the elbow, trunk and face (limited can have some facial involvement). Limited scleroderma has a 90% 5-year survival, diffuse scleroderma 70% 5-year survival. In the past, renal crises were the most common cause of death in patients with scleroderma. Aggressive treatment with blood pressure lowering drugs, particularly those known as ACE inhibitors, is proving to be successful in reducing this risk.

Anti-dsDNA and Rh factor are indicative of SLE an RA/Sjogrens/others, but there is no evidence of arthritis or cutaneous rashes. Anti-Jo-1 antibody is a marker of polymyositis.



Next question >

## Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

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- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis



Next question >



# **Textbooks** High-yield textbook Extended textbook Links DermNet NZ Systemic sclerosis Suggest link Report broken link Media Scleroderma Townsend Teaching - YouTube 4 Q 0 SCLERODERMA Scleroderma ტ 4 ♀ 1 Osmosis - YouTube SYSTEMIC SCLEROSIS Systemic Sclerosis and Scleroderma Zero to Finals - YouTube Report broken media

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Question 89 of 178





A 21-year-old male presents to his GP complaining of muscle cramps that prevent him from competing in his local park 5 km race. He has always had muscle pains when warming up with exercise but these gradually diminish after 20 minutes. There was no weakness and no abnormalities on neurological exam.

Creatinine kinase was elevated at 1215 IU/L and myoglobinuria was noted on urinalysis. The electromyography (EMG) demonstrated myotonic discharges and fibrillations.

What is the likely diagnosis?

Hypokalaemic periodic paralysis	
Von Gierke disease	
McArdle disease	
Pompe disease	
Gaucher disease	

Submit answer

Reference ranges ✓

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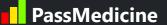
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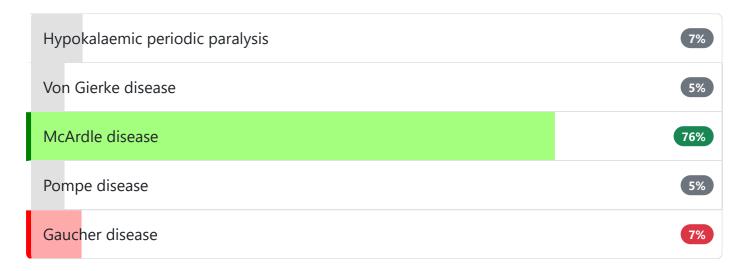
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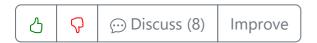
What is the likely diagnosis?



McArdle disease (also known as myophosphorylase deficiency or Glycogen storage disease V) often presents in adolescence with exercise intolerance, cramps and weakness. Unfortunately it is often misdiagnosed as chronic fatigue syndrome. Inheritance is autosomal recessive. The diagnostic clues are:

- No rise in venous blood lactate on exercise
- Muscle biopsy demonstrates elevated glycose concentration and muscle phosphorylase deficiency

Investigations usually reveal an elevated CK and myoglobinuria. Diagnosis is confirmed with forearm muscle exercise testing or genetic testing. Management involves avoidance of low carbohydrate diets and low intensity aerobic exercise.



#### McArdle's disease

#### Overview

- autosomal recessive type V glycogen storage disease
- caused by myophosphorylase deficiency
- this causes decreased muscle glycogenolysis

#### **Features**

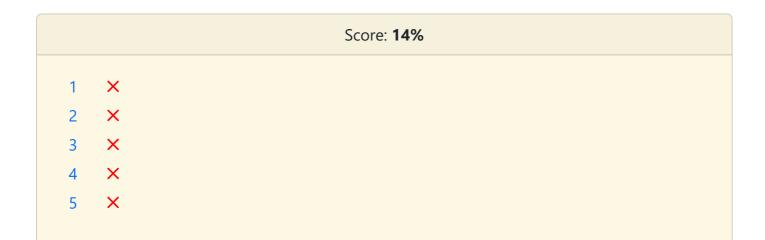
- muscle pain and stiffness following exercise
- muscle cramps
- rhabdomyolysis & myoglobinuria
- low lactate levels during exercise



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Question 90 of 178





A 26-year-old female presented with lethargy, intermittent low grade fever, bilateral wrist pain, pleuritic type chest pain and an erythematous scaly rash perinasally. She had no significant medical history and her family history was significant for an aunt with autoimmune hepatitis and a brother with type 1 diabetes and pernicious anaemia.

Examination reveals a well young lady. Blood pressure 110/78mmHg and pulse 76/min. Abdominal and cardiovascular examination were normal. There was dullness to percussion over both lung bases. She had swelling and tenderness of her wrists. A malar rash on a background of moderate facial acne with some scaring were noted. She gives a history of acne which has improved ever since she started taking minocycline prescribed by her GP 3 months ago. She is also on oral contraceptives for irregular periods.

#### Investigations:

Hb	10.3 g/dl
MCV	79 fl
Platelets	256 * 10 <sup>9</sup> /l
WBC	7 * 10 <sup>9</sup> /l
Creatinine	88 umol/L
Na+	140 mmol/L
K+	3.6 mmol/L
ANA	Positive
Anti dsDNA	Negative
Chest X-ray	Blunting of costophrenic angles bilaterally
Complements C3 & C4	normal

What investigation is most likely to confirm the suspected diagnosis?

Serum rheumatoid factor, anti Ro, anti La	
Renal biopsy	
Wrist X-ray	
Anti histone antibody	
Repeat serum anti dsDNA to rule out lab error	

## Submit answer

Reference ranges  $\checkmark$ 

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Question 90 of 178







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Na+	140 mmol/L
K+	3.6 mmol/L
ANA	Positive
Anti dsDNA	Negative
Chest X-ray	Blunting of costophrenic angles bilaterally
Complements C3 & C4	normal

What investigation is most likely to confirm the suspected diagnosis?

Serum rheumatoid factor, anti Ro, anti La	11%
Renal biopsy	4%
Wrist X-ray	1%
Anti histone antibody	78%
Repeat serum anti dsDNA to rule out lab error	5%

Answer: Serum anti histone antibodies.

This is a typical case of drug induced lupus (DIL). Patients give symptoms similar to systemic lupus erythematosus (SLE) but they are unlikely to have renal and CNS involvement.

Auto antibody screening would reveal positive anti histone antibodies and negative anti-dsDNA antibodies. ANA positivity is common.

Some common causes of drug induced lupus include minocycline, isoniazid, hydralazine, procainamide. Minocycline in particular uniquely causes a repeat episode of drug induced lupus when patient is re-challenged.

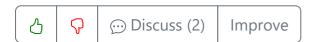
The main treatment is stopping the offending medication.

Features differentiating drug induced lupus from SLE:

- SLE is more common in patients of African descent.
- Drug induced lupus is more common in patients of European descent.
- Renal and CNS involvements are rare in drug induced lupus whereas they are features of systemic lupus erythematosus.
- Though anti histone antibodies can be found in up to 50% of patients with SLE, they are much more likely to be associated with DIL, whilst anti ds-DNA is more specific to systemic lupus erythematosus.
- It is also important to note that complement levels are normal in DIL but low in SLE.

Minocycline induced autoimmune hepatitis and systemic lupus erythematosus-like syndrome BMJ 1996; 312 doi: http://dx.doi.org/10.1136/bmj.312.7024.169 (Published 20 January 1996) http://www.bmj.com/content/312/7024/169

http://emedicine.medscape.com/article/1065086-workup#c7



Next question >

### Drug-induced lupus \*

In drug-induced lupus not all the typical features of systemic lupus erythematosus are seen, with renal and nervous system involvement being unusual. It usually resolves on stopping the drug.

#### **Features**

- arthralgia
- myalgia

- skin (e.g. malar rash) and pulmonary involvement (e.g. pleurisy) are common
- ANA positive in 100%, dsDNA negative
- anti-histone antibodies are found in 80-90%
- anti-Ro, anti-Smith positive in around 5%



#### Most common causes

- procainamide
- hydralazine

#### Less common causes

- isoniazid
- minocycline
- phenytoin



Next question >



#### **Textbooks**

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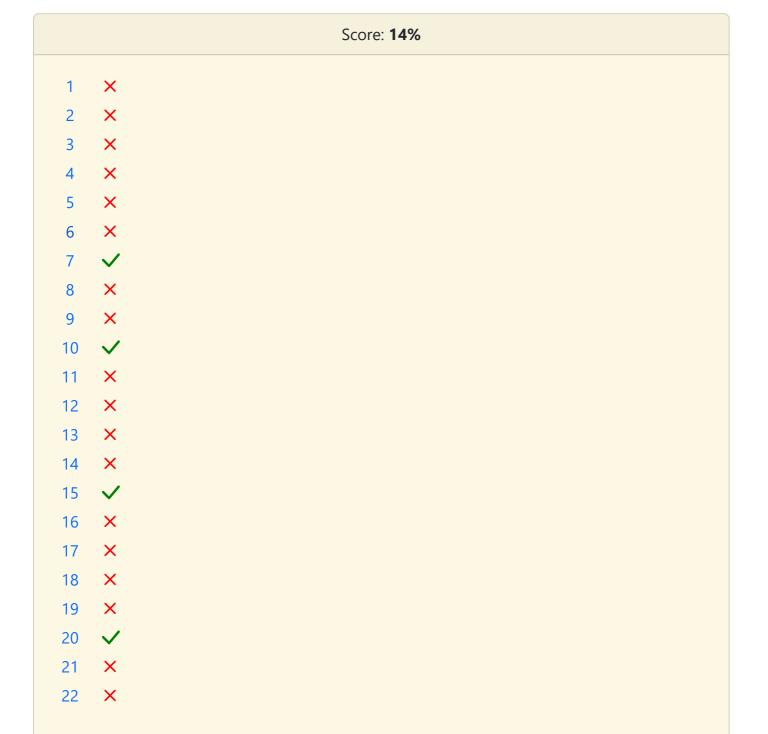
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Drug-induced lupus erythematosus

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Question 91 of 178





A 70-year-old man presents with a two-year history of stiffness and pain in his hands. The stiffness is worse in the mornings and tends to gradually ease after around an hour. On examination you note synovitis and swelling affecting the distal interphalangeal joints in both hands.

An x-ray is requested and shown below:



© Image used on license from Radiopaedia

#### What is the most likely diagnosis?

Osteoarthritis	
Myeloma	
Gout	
Rheumatoid arthritis	
Psoriatic arthritis	

Reference ranges  $\vee$ 

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Question 91 of 178



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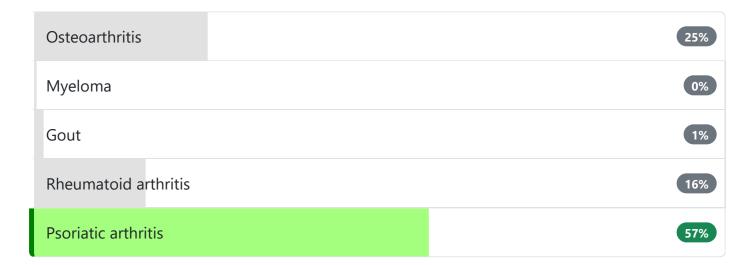
A 70-year-old man presents with a two-year history of stiffness and pain in his hands. The stiffness is worse in the mornings and tends to gradually ease after around an hour. On examination you note synovitis and swelling affecting the distal interphalangeal joints in both hands.

An x-ray is requested and shown below:



© Image used on license from Radiopaedia

### What is the most likely diagnosis?



Note that the DIPs are predominately affected, rather than the MCPs and PIPs as would be seen with rheumatoid. Extensive juxta-articular periostitis is seen in the DIPs but the changes have not yet progressed to the classic 'pencil-in-cup' changes that are often seen with psoriatic arthritis.

Next question >

# Psoriatic arthropathy \*

Psoriatic arthropathy is an inflammatory arthritis associated with psoriasis and is classed as one of the seronegative spondyloarthropathies. It correlates poorly with cutaneous psoriasis and often precedes the development of skin lesions. Around 10-20% of patients with skin lesions develop an arthropathy with males and females being equally affected.

#### **Presentation**

#### **Patterns**

- symmetric polyarthritis
  - very similar to rheumatoid arthritis
  - o 30-40% of cases, most common type
- asymmetrical oligoarthritis: typically affects hands and feet (20-30%)
  - until recently it was thought asymmetrical oligoarthritis was the most common type,
     based on data from the original 1973 Moll and Wright paper. Please see the link for a comparison of more recent studies
- sacroiliitis
- DIP joint disease (10%)
- arthritis mutilans (severe deformity fingers/hand, 'telescoping fingers')

#### Other signs

- psoriatic skin lesions
- periarticular disease tenosynovitis and soft tissue inflammation resulting in:
  - enthesitis: inflammation at the site of tendon and ligament insertion e.g. Achilles tendonitis, plantar fascitis
  - tenosynovitis: typically of the flexor tendons of the hands
  - dactylitis: diffuse swelling of a finger or toe
- nail changes
  - pitting
  - o onycholysis

## Investigation and management

#### X-ray

 often have the unusual combination of coexistence of erosive changes and new bone formation

- periostitis
- 'pencil-in-cup' appearance

#### Management

- should be managed by a rheumatologist
- treatment is similar to that of rheumatoid arthritis (RA). However, the following differences are noted:
  - mild peripheral arthritis/mild axial disease may be treated with 'just' an NSAID, rather than all patients being on disease-modifying therapy as with RA
  - o if more moderate/severe disease then methotrexate is typically used as in RA
  - use of monoclonal antibodies such as ustekinumab (targets both IL-12 and IL-23) and secukinumab (targets IL-17)
  - o apremilast: phosphodiesterase type-4 (PDE4) inhibitor → suppression of proinflammatory mediator synthesis and promotion of anti-inflammatory mediators
  - o has a better prognosis than RA











Next question >

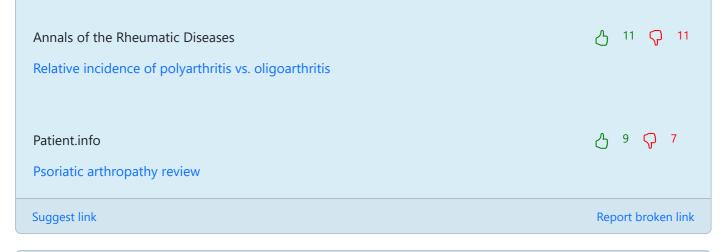


## **Textbooks**

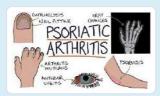
High-yield textbook

Extended textbook

#### Links



### Media



#### Psoriatic arthritis

Zero To Finals - YouTube 6 7 7 1





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A 60-year-old man is referred to the rheumatology clinic with progressive pains in his hands. His past medical history includes hypertension for which he takes ramipril and indapamide. The referral letter includes the following bloods taken during a recent flare:

CRP	54 mg/l
Rheumatoid factor	negative
Adj calcium	2.51 mmol/l

## An x-ray is taken:



## What is the most likely diagnosis?

Metastatic prostate cancer	
Rheumatoid arthritis	
Osteoarthritis	
Gout	
Primary hyperparathryoidism	

Submit answer

Reference ranges  $\vee$ 

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### Score: **18%** X 1 2 X 3 X 4 X 5 X X 6 7 **/** 8 X X 9 10 11 X 12 X 13 X X 14 15 **/** X 16 X 17 X 18 19 X 20 X 21 22 X 23 X 24 X 25 **/** 26 27 X 28 X 29 30 X

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A 60-year-old man is referred to the rheumatology clinic with progressive pains in his hands. His past medical history includes hypertension for which he takes ramipril and indapamide. The referral letter includes the following bloods taken during a recent flare:

CRP	54 mg/l
Rheumatoid factor	negative
Adj calcium	2.51 mmol/l

### An x-ray is taken:



### What is the most likely diagnosis?



The x-ray shows multiple periarticular erosions bilaterally with adjacent large soft tissue masses and relatively preserved joint spaces. In the right hand, these findings are most prominent at the

1st interphalangeal, 2nd-4th proximal interphalangeal, 1st-3rd metacarpohalangeal and carpometacarpal joints. In the left hand, the findings are most prominent at the ulnar styloid, scapholunate joint, first and fifth carpometacarpal joints, second and fifth metacarpophalangeal joints and 1st interphalangeal joint. These findings are consistent with gout.

Indapamide is a risk factor for gout.

A normal calcium goes against primary hyperparathyroidism.



Next question >

## Gout: features \*

#### **Features**

Gout is a form of inflammatory arthritis. Patients typically have episodes lasting several days when their gout flares and are often symptom-free between episodes. The acute episodes typically develop maximal intensity with 12 hours. The main features it presents with are:

- pain: this is often very significant
- swelling
- erythema

Around 70% of first presentations affect the 1st metatarsophalangeal (MTP) joint. Attacks of gout affecting this area were historically called podagra. Other commonly affected joints include:

- ankle
- wrist
- knee

If untreated repeated acute episodes of gout can damage the joints resulting in a more chronic joint problem.

## Investigations

Uric acid

- NICE recommends measuring uric acid levels in suspected gout (i.e. in the acute setting)
  - o a uric acid level ≥ 360 umol/L is seen as supporting a diagnosis
  - if uric acid level < 360 umol/L during a flare and gout is strongly suspected, repeat the uric acid level measurement at least 2 weeks after the flare has settled

## Synovial fluid analysis

• needle shaped negatively birefringent monosodium urate crystals under polarised light

### Radiological features of gout include:

- joint effusion is an early sign
- well-defined 'punched-out' erosions with sclerotic margins in a juxta-articular distribution, often with overhanging edges
- relative preservation of joint space until late disease
- eccentric erosions
- no periarticular osteopenia (in contrast to rheumatoid arthritis)
- soft tissue tophi may be seen







Next question >













Links

Gout - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube

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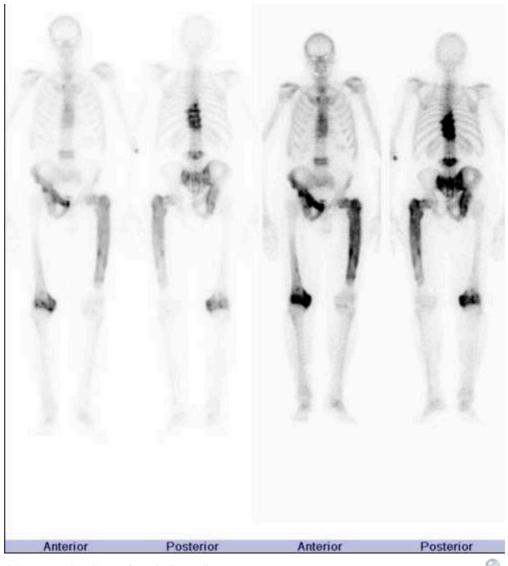
Question 93 of 178





A 76-year-old is investigated for persistent and progressive pain in his back and left hip. This is no longer responding to standard analgesia and has resulted in him taking regular modified-release morphine sulphate. Standard plain films of his left hip did not show changes consistent with osteoarthritis.

An isotope bone scan is therefore ordered to investigate his pain further:



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### What is the most likely cause of his pain?

Multiple myeloma	
Metastatic prostate cancer	
Paget's disease of the bone	

Osteoporosis	
Ankylosing spondylitis	

## Submit answer

Reference ranges ✓

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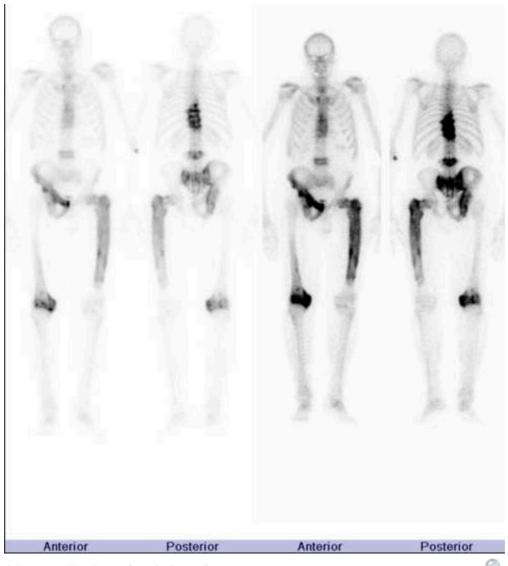


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A 76-year-old is investigated for persistent and progressive pain in his back and left hip. This is no longer responding to standard analgesia and has resulted in him taking regular modified-release morphine sulphate. Standard plain films of his left hip did not show changes consistent with osteoarthritis.

An isotope bone scan is therefore ordered to investigate his pain further:



© Image used on license from Radiopaedia



### What is the most likely cause of his pain?

Multiple myeloma	15%
Metastatic prostate cancer	25%
Paget's disease of the bone	55%

The bone scan demonstrates intense uptake involving several lower thoracic vertebrae, L3, right hemipelvis, sacrum, left proximal femur and right knee. There is expansion and bowing of the involved femur. These changes are typical of Paget's disease.

Isotope bone scans are not useful for asssessing multiple myeloma or osteoporosis.



Next question >

# Paget's disease of the bone \*

Paget's disease is a disease of increased but uncontrolled bone turnover. It is thought to be primarily a disorder of osteoclasts, with excessive osteoclastic resorption followed by increased osteoblastic activity. Paget's disease is common (UK prevalence 5%) but symptomatic in only 1 in 20 patients. The skull, spine/pelvis, and long bones of the lower extremities are most commonly affected.

#### Predisposing factors

- increasing age
- male sex
- northern latitude
- family history

Clinical features - only 5% of patients are symptomatic

- the stereotypical presentation is an older male with bone pain and an isolated raised ALP
- bone pain (e.g. pelvis, lumbar spine, femur)
- classical, untreated features: bowing of tibia, bossing of skull

#### Investigations

- bloods
  - raised alkaline phosphatase (ALP)
  - calcium and phosphate are typically normal. Hypercalcaemia may occasionally occur with prolonged immobilisation
- other markers of bone turnover include
  - procollagen type I N-terminal propeptide (PINP)
  - serum C-telopeptide (CTx)

- o urinary N-telopeptide (NTx)
- o urinary hydroxyproline
- x-rays
  - o osteolysis in early disease → mixed lytic/sclerotic lesions later
  - o skull x-ray: thickened vault, osteoporosis circumscripta
- bone scintigraphy
  - o increased uptake is seen focally at the sites of active bone lesions

### Management

- indications for treatment include
  - o bone pain
  - skull or long bone deformity
  - o fracture
  - o periarticular Paget's
- bisphosphonate (either oral risedronate or IV zoledronate)
- calcitonin is less commonly used now

### Complications

- deafness (cranial nerve entrapment)
- bone sarcoma (1% if affected for > 10 years)
- fractures
- skull thickening
- high-output cardiac failure



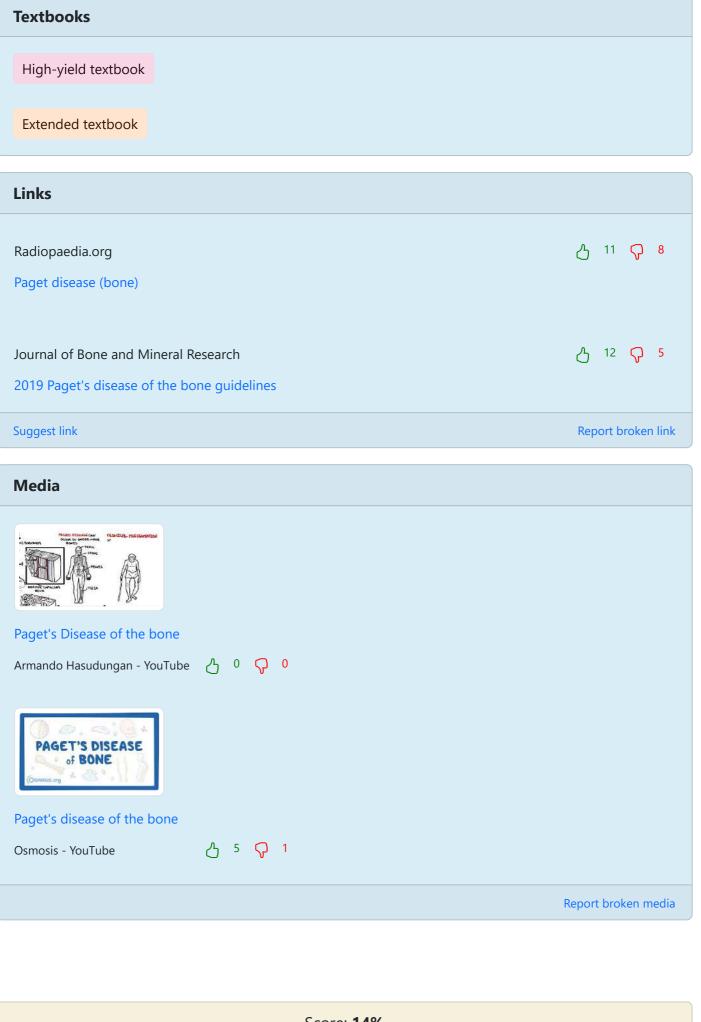






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A 48-year-old woman presents with progressively worsening pain in the right shoulder over the past few weeks. She is generally fit and well but smokes 20 cigarettes/day.

On examination there is diffuse mild tenderness over the lateral aspect of the right shoulder. The pain is recreated when abducting the should to around 70-80 degrees.

A shoulder x-ray is requested:



What is the most likely diagnosis?

Pancoast tumour	
Supraspinatus tendonitis	
Adhesive capsulitis	
Humeral head fracture	
Avascular necrosis	

Submit answer

Reference ranges ✓

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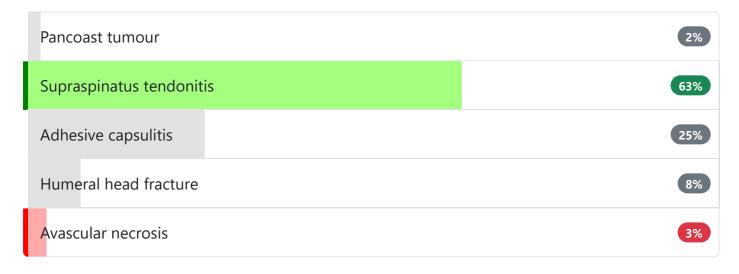
A 48-year-old woman presents with progressively worsening pain in the right shoulder over the past few weeks. She is generally fit and well but smokes 20 cigarettes/day.

On examination there is diffuse mild tenderness over the lateral aspect of the right shoulder. The pain is recreated when abducting the should to around 70-80 degrees.

A shoulder x-ray is requested:



## What is the most likely diagnosis?



The x-ray shows calcification of the supraspinatus tendon consistent with prolonged inflammation. On examination the patient exhibits the classical 'painful arc' associated with this condition.

Next question >

# Shoulder problems \*

The table below summarises the key features of common shoulder problems:

Condition	Notes
Adhesive capsulitis (frozen shoulder)	Common in middle-age and diabetics Characterised by painful, stiff movement Limited movement in all directions, with loss of external rotation and abduction in about 50% of patients
Supraspinatus tendonitis (Subacromial impingement, painful arc)	Rotator cuff injury Painful arc of abduction between 60 and 120 degrees Tenderness over anterior acromion



Next question >





### Media



#### **Shoulder Examination**

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Question 95 of 178





A 37-year-old woman presents with left-sided pain shoulder pain. This has been present for around 6 months and is described variably as a 'toothache' or an 'electric shock' sensation which extends from her neck to the elbow.

On examination there is reduced power when abducting the left shoulder and reduced sensation to light touch just inferior to the deltoid muscle. The biceps reflex on the left side is absent. Pain and temperature sensation are normal in the left arm. Examination of the right arm is normal.

### An MRI neck is requested:



© Image used on license from Radionaedia



## What is the most likely diagnosis?

Bony metastases	
Brown-Sequard syndrome	
Arnold-Chiari malformation	

Syringomyelia	
Cervical disc prolapse	

# Submit answer

Reference ranges ✓

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Question 95 of 178



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A 37-year-old woman presents with left-sided pain shoulder pain. This has been present for around 6 months and is described variably as a 'toothache' or an 'electric shock' sensation which extends from her neck to the elbow.

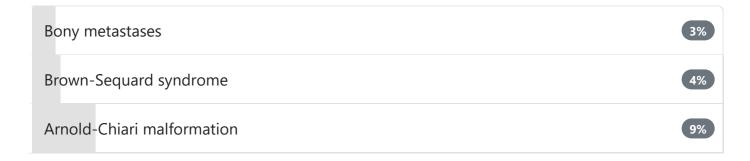
On examination there is reduced power when abducting the left shoulder and reduced sensation to light touch just inferior to the deltoid muscle. The biceps reflex on the left side is absent. Pain and temperature sensation are normal in the left arm. Examination of the right arm is normal.

## An MRI neck is requested:



© Image used on license from Radiopaedia

## What is the most likely diagnosis?



# Cervical disc prolapse

64%

This patient has signs consistent with a C5/6 myelopathy. Note the very large extruded disc at C5/6. There is also fusion of C2 and C3 with adjacent segment degeneration and cord volume loss suggesting myelomalacia.



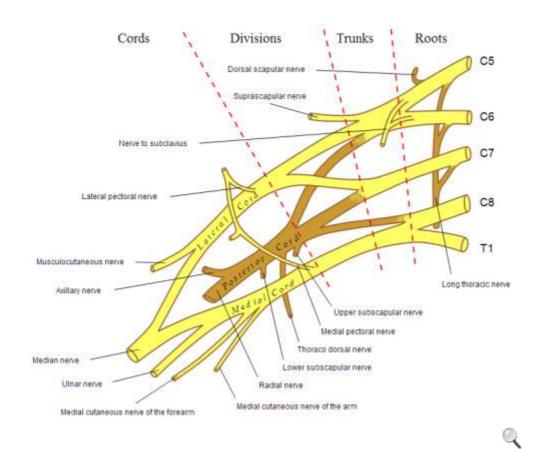
Next question >

# Upper limb anatomy \*

The information below contains selected facts which commonly appear in examinations:

Nerve	Motor	Sensory	Typical mechanism of injury & notes
Musculocutaneous nerve (C5-C7)	Elbow flexion (supplies biceps brachii) and supination	Lateral part of the forearm	Isolated injury rare - usually injured as part of brachial plexus injury
Axillary nerve (C5,C6)	Shoulder abduction (deltoid muscle)	Inferior region of the deltoid muscle	Humeral neck fracture/dislocation  Results in flattened deltoid
Radial nerve (C5-C8)	Extension (forearm, wrist, fingers, thumb)	Small area between the dorsal aspect of the 1st and 2nd metacarpals	Humeral midshaft fracture  Palsy results in wrist drop
Median nerve (C6, C8, T1)	LOAF* muscles  Features depend on the site of the lesion:  • wrist: paralysis of thenar muscles,	Palmar aspect of lateral 3½ fingers	Wrist lesion → carpal tunnel syndrome

Nerve	Motor	Sensory	Typical mechanism of injury & notes
	opponens pollicis elbow: loss of pronation of forearm and weak wrist flexion		
Ulnar nerve (C8, T1)	Intrinsic hand muscles except LOAF* Wrist flexion	Medial 1½ fingers	Medial epicondyle fracture  Damage may result in a 'claw hand'
Long thoracic nerve (C5-C7)	Serratus anterior		Often during sport e.g. following a blow to the ribs. Also possible complication of mastectomy  Damage results in a winged scapula



### Erb-Duchenne palsy ('waiter's tip')

- due to damage of the upper trunk of the brachial plexus (C5,C6)
- may be secondary to shoulder dystocia during birth
- the arm hangs by the side and is internally rotated, elbow extended

### Klumpke injury

- due to damage of the lower trunk of the brachial plexus (C8, T1)
- as above, may be secondary to shoulder dystocia during birth. Also may be caused by a sudden upward jerk of the hand
- associated with Horner's syndrome

#### \*LOAF muscles

- Lateral two lumbricals
- Opponens pollis
- Abductor pollis brevis
- Flexor pollis brevis



Next question >

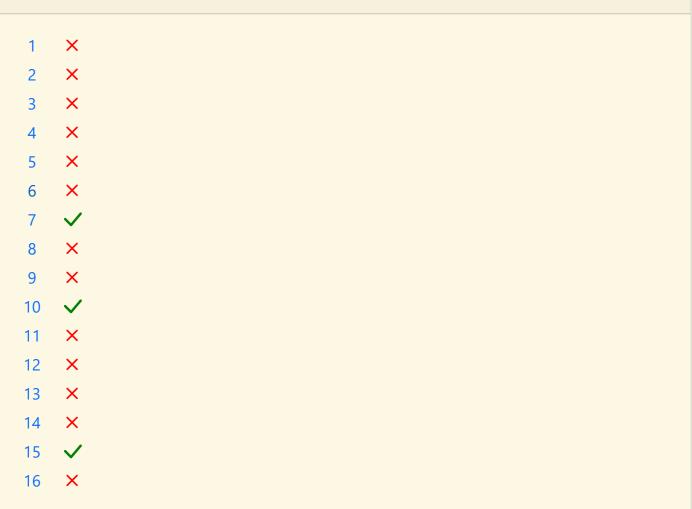




## Media



# **Upper Extremity Nerve Injuries** DirtyUSMLE - YouTube KLUMPKE'S PALSY & THORACIC OUTLET SYNDROME Klumpke's palsy and thoracic outlet syndrome ტ 0 ♀ 0 Osmosis - YouTube Learn The Bones of the Hand & Wrist In 2 Minutes (With Fractures) ტ 0 ♀ 0 Rhesus Medicine - YouTube Report broken media Score: **14%** X 1 X 2



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Question 96 of 178





A 40-year-old man presents to the Emergency Department with pain in his left foot. He thinks this may have been triggered by dropping a heavy box on it at work a few days ago. He is known to have type 2 diabetes mellitus which is managed with metformin. On examination there is erythema, tenderness and swelling in the distal, medial aspect of the left foot.

## Bloods show the following:

Hb	145 g/l
Platelets	311 * 10 <sup>9</sup> /l
WBC	6.3 * 10 <sup>9</sup> /I
CRP	56 mg/l

## An x-ray is requested:



© Image used on license from Radiopaedia



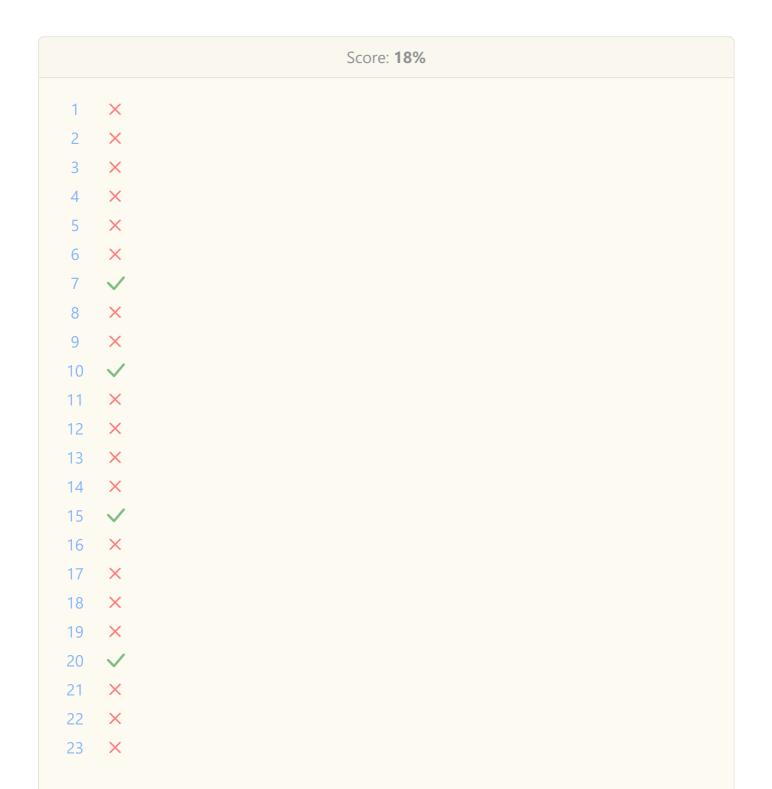
## What is the most appropriate management?

Naproxen	
Allopurinol	

Below-the-knee plaster cast	
Intravenous antibiotics	
Supportive stocking e.g. TubiGrip	

# Submit answer

Reference ranges ✓



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Question 96 of 178



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WBC	6.3 * 10 <sup>9</sup> /l
CRP	56 mg/l

## An x-ray is requested:



© Image used on license from Radiopaedia

## What is the most appropriate management?





The x-ray demonstrates juxta-articular erosive changes around the 1st MTP joint with overhanging edges and associated with a moderate soft tissue swelling. The joint space is maintained. These findings are consistent with gout.

Allopurinol should not be used in the acute phase of gout. NSAIDs such as naproxen are generally used first-line, if there are no contraindications.



Next question >

## Gout: features \*

#### **Features**

Gout is a form of inflammatory arthritis. Patients typically have episodes lasting several days when their gout flares and are often symptom-free between episodes. The acute episodes typically develop maximal intensity with 12 hours. The main features it presents with are:

- pain: this is often very significant
- swelling
- erythema

Around 70% of first presentations affect the 1st metatarsophalangeal (MTP) joint. Attacks of gout affecting this area were historically called podagra. Other commonly affected joints include:

- ankle
- wrist
- knee

If untreated repeated acute episodes of gout can damage the joints resulting in a more chronic joint problem.

# Investigations

Uric acid

- NICE recommends measuring uric acid levels in suspected gout (i.e. in the acute setting)
  - o a uric acid level ≥ 360 umol/L is seen as supporting a diagnosis
  - if uric acid level < 360 umol/L during a flare and gout is strongly suspected, repeat the uric acid level measurement at least 2 weeks after the flare has settled

#### Synovial fluid analysis

• needle shaped negatively birefringent monosodium urate crystals under polarised light

### Radiological features of gout include:

- joint effusion is an early sign
- well-defined 'punched-out' erosions with sclerotic margins in a juxta-articular distribution, often with overhanging edges
- relative preservation of joint space until late disease
- eccentric erosions
- no periarticular osteopenia (in contrast to rheumatoid arthritis)
- soft tissue tophi may be seen







Next question >

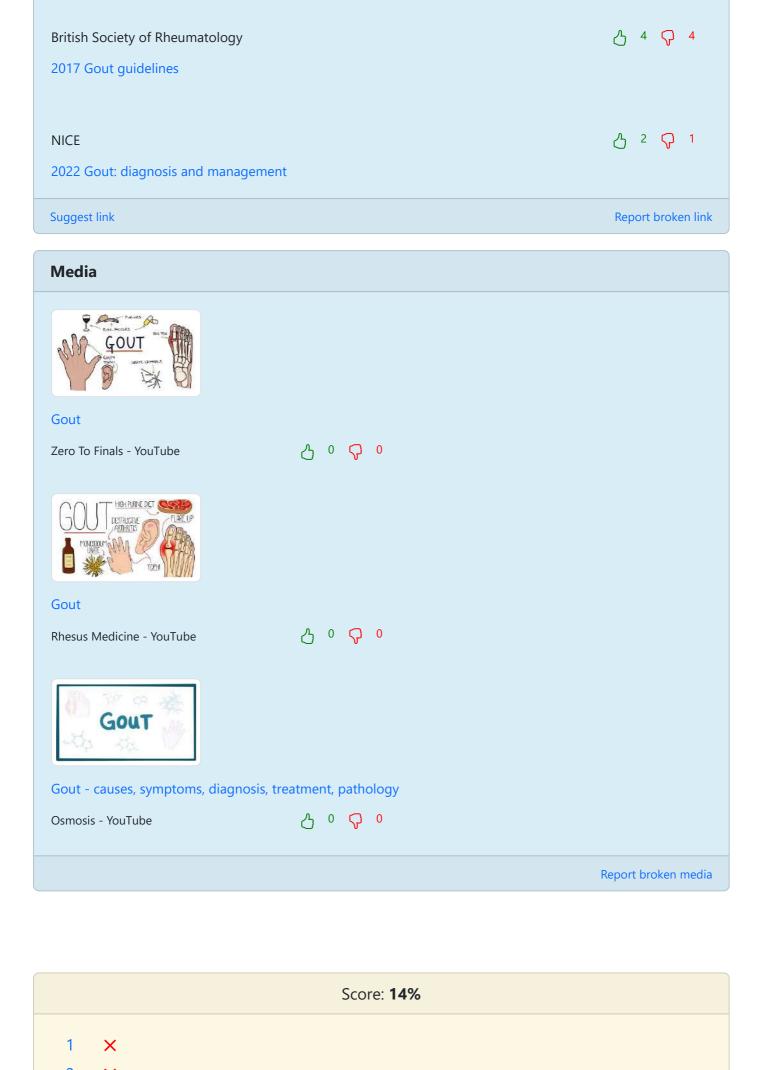


# **Textbooks**

High-yield textbook

Extended textbook

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Question 97 of 178





A 71-year-old man with a history of chronic obstructive pulmonary disease (COPD) is investigated for back pain. Over the past 10 years he has had numerous admissions for infective exacerbations of COPD and currently uses long-term oxygen therapy. The pain came on suddenly whilst he was at his local supermarket.

# A MRI scan is requested:



© Image used on license from Radiopaedia

## What is the most likely underlying cause of the back pain?

Osteomyelitis	
Multiple myeloma	
Pott's disease	
Metastatic lung cancer	

# Submit answer

Reference ranges  $\checkmark$ 

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Question 97 of 178







A 71-year-old man with a history of chronic obstructive pulmonary disease (COPD) is investigated for back pain. Over the past 10 years he has had numerous admissions for infective exacerbations of COPD and currently uses long-term oxygen therapy. The pain came on suddenly whilst he was at his local supermarket.

## A MRI scan is requested:



© Image used on license from Radiopaedia

## What is the most likely underlying cause of the back pain?

Osteomyelitis	6%
Multiple myeloma	3%
Pott's disease	11%
Metastatic lung cancer	10%

The MRI shows osteoporotic fractures of the 10th and 12th thoracic vertebrae. This is likely to have been caused by repeated courses of steroids to treat exacerbations of COPD.



Next question >

# Osteoporosis: management \*

The National Osteoporosis Guideline Group (NOGG) updated their guidelines in 2021. NICE guidelines also have a section on the management of osteoporosis, largely based on the NOGG guidelines. Remember that osteoporosis is usually asymptomatic until a fracture occurs. When thinking about osteoporosis management it is useful to think about a number of potential clinical scenarios:

- a patient who has been identified as being at high risk of a fragility fracture based on a QFracture or FRAX score (please see the textbook entry on 'Osteoporosis: assessing risk')
- a patient who is about to start treatment that puts them at significant risk of developing osteoporosis the most common example is longer-term glucocorticoids
- a patient who has just had a fragility fracture e.g. a symptomatic osteoporotic vertebral fracture

# **General management points**

General points about the management of all patients

- all patients who are at risk of osteoporosis or have osteoporosis should be given advice regarding:
  - lifestyle changes: a healthy, balanced diet, moderation of alcohol consumption and avoidance of smoking
  - a sufficient dietary calcium and vitamin D intake: supplementation should be offered to all women unless the clinician is confident they have adequate calcium intake and are vitamin D replete
  - encourage a combination of regular weight-bearing and muscle strengthening exercise
- secondary causes of osteoporosis should be considered and treated
  - e.g. hypogonadism in women or men e.g. hormone replacement therapy for premature menopause
- bisphosphonates are the first-line drug treatment for patients at risk of fragility fractures
  - oral bisphosphonates such as alendronate and risedronate are typically first-line. These are often taken weekly are need taking in a particular way to minimise the risk of oesophageal side-effects

- however, the NOGG recommend IV zoledronate as the first-line treatment following a hip fracture. This is given yearly
- denosumab is generally used as a second-line treatment
- other possible treatment options include:
  - o strontium ranelate
  - raloxifene
  - o teriparatide
  - romosozumab

### Clinical scenarios

### Fragility risk fracture assessment

- if a patient is deemed high-risk based on a QFracture or FRAX score they should have a DEXA scan to assess bone mineral density (BMD)
  - the BMD threshold for defining osteoporosis is a T-score of 2.5 SD or below
  - o some patients may not be suitable for BMD assessment due to frailty etc.
- general osteoporosis management as above
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

Postmenopausal women, and men age ≥50, who are treated with oral glucocorticoids:

- if starting ≥7.5 mg/day prednisolone or equivalent for the next 3 months, start bone protective treatment at the same time
- general osteoporosis management as above
- don't wait for a DEXA scan before starting treatment
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

A postmenopausal woman, or a man age ≥50 has a symptomatic osteoporotic vertebral fracture:

- general osteoporosis management as above
- start treatment straight away oral bisphosphonates are used first-line e.g. alendronate or risedronate

### Hip fracture in older adults

- in older adults a hip fracture is a manifestation of osteoporosis
  - o following a fragility fracture in women ≥ 75 years, a DEXA scan is not necessary to diagnose osteoporosis and hence commence a bisphosphonate
  - BMD should be measured, but this acts as a baseline rather than determining whether treatment should be given
- bisphosphonates should be given first-line
  - NOGG recommends IV zoledronate but local guidelines may vary and oral bisphosphonates are often used

Plan to prescribe oral bisphosphonates for at least 5 years, or intravenous bisphosphonates for at least 3 years and then re-assess fracture risk.

### Supplementary notes on treatment

### Bisphosphonates

- bisphosphonates bind to hydroxyapatite in bone, inhibiting osteoclast-mediated bone resorption
- common side effects include gastrointestinal discomfort, oesophagitis, and hypocalcaemia. Atypical femoral fractures and osteonecrosis of the jaw are rare but serious risks.
- available in oral and intravenous formulations. Oral bisphosphonates should be taken with a full glass of water, on an empty stomach, and the patient should remain upright for at least 30 minutes afterwards.

#### Denosumab

- human monoclonal antibody that inhibits RANK ligand, which in turn inhibits the maturation of osteoclasts
- also used for cancer patients with bone metastases to reduce skeletal-related events.
- given as a single subcutaneous injection every 6 months

### Raloxifene

- selective oestrogen receptor modulator (SERM)
- has been shown to prevent bone loss and to reduce the risk of vertebral fractures, but has not yet been shown to reduce the risk of non-vertebral fractures
- has been shown to increase bone density in the spine and proximal femur
- may worsen menopausal symptoms
- increased risk of thromboembolic events
- may decrease the risk of breast cancer

### Strontium ranelate

- 'dual action bone agent' increases deposition of new bone by osteoblasts (promotes differentiation of pre-osteoblast to osteoblast) and reduces the resorption of bone by inhibiting osteoclasts
- concerns regarding the safety profile of strontium have been raised recently. It should only be prescribed by a specialist in secondary care
- due to these concerns the European Medicines Agency in 2014 said it should only be used by people for whom there are no other treatments for osteoporosis
- increased risk of cardiovascular events: any history of cardiovascular disease or significant risk of cardiovascular disease is a contraindication
- increased risk of thromboembolic events: a Drug Safety Update in 2012 recommended it is not used in patients with a history of venous thromboembolism
- may cause serious skin reactions such as Stevens Johnson syndrome

### Teriparatide

- recombinant form of parathyroid hormone
- very effective at increasing bone mineral density but role in the management of osteoporosis yet to be clearly defined

#### Romosozumab

- a monoclonal antibody that inhibits sclerostin, thereby increasing bone formation and decreasing bone resorption
- this dual action significantly improves bone density and reduces fracture risk.



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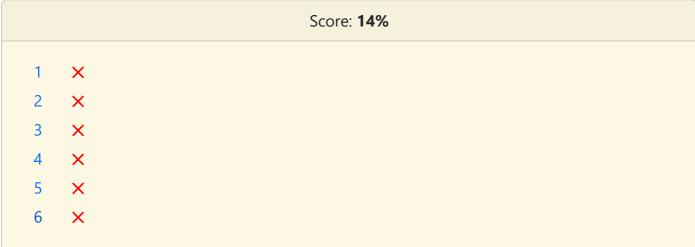
MRI showing osteoporotic fractures of the 10th and 12th thoracic vertebrae.



Next question >



## **Textbooks** High-yield textbook Extended textbook Links 占 9 ♀ 11 The National Osteoporosis Guideline Group 2021 Clinical guideline for the prevention and treatment of osteoporosis NICE 2008 Osteoporosis: secondary prevention Suggest link Report broken link Media SECEDIOS/AKENHA VEOLOG/AKENHA Osteoporosis pharmacology, prevention and treatment 少 5 ♀ 0 Armando Hasudungan - YouTube Report broken media



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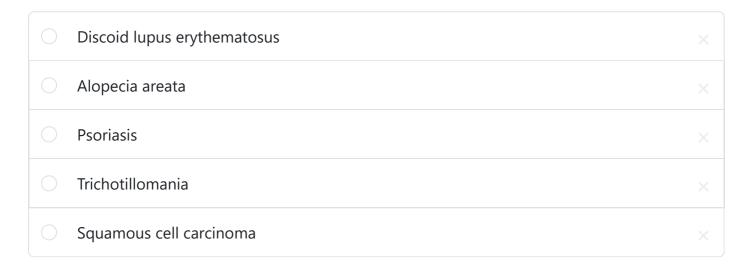




A 35-year-old woman presents with alopecia. For the past few months she has noticed some 'scaly' patches on her scalp. Once healed they normally leave a scar and no hair seems to grow back. Her scalp has the following appearance:



What is the most likely diagnosis?



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A 35-year-old woman presents with alopecia. For the past few months she has noticed some 'scaly' patches on her scalp. Once healed they normally leave a scar and no hair seems to grow back. Her scalp has the following appearance:



### What is the most likely diagnosis?



Remember that alopecia may be divided into scarring (destruction of hair follicle) and non-scarring (preservation of hair follicle):

### Scarring alopecia

- trauma, burns
- radiotherapy
- lichen planus
- discoid lupus
- tinea capitis (if untreated)

### Non-scarring alopecia

- male-pattern baldness
- drugs: cytotoxic drugs, carbimazole, heparin, oral contraceptive pill, colchicine
- nutritional: iron and zinc deficiency
- autoimmune: alopecia areata
- telogen effluvium (hair loss following stressful period e.g. surgery)
- trichotillomania

Even if you are not familiar with the appearance of discoid lupus erythematosus (along with most non-dermatologists) this leaves it as the only possible answer.



Next question >

### Discoid lupus erythematosus \*

Discoid lupus erythematosus is a benign disorder generally seen in younger females. It very rarely progresses to systemic lupus erythematosus (in less than 5% of cases). Discoid lupus erythematosus is characterised by follicular keratin plugs and is thought to be autoimmune in aetiology

#### **Features**

- erythematous, raised rash, sometimes scaly
- may be photosensitive
- more common on face, neck, ears and scalp
- lesions heal with atrophy, scarring (may cause scarring alopecia), and pigmentation

### Management

- topical steroid cream
- oral antimalarials may be used second-line e.g. hydroxychloroquine
- avoid sun exposure





Next question >







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A 67-year-old woman presents with a rash. For the past two weeks she has felt tired and 'achey'. She also has a dry cough and some pleuritic chest pain. She is most concerned however with a new rash on her face:



Which drug is most likely to cause this presentation?

Procainamide	
Digoxin	×
Sodium valproate	×
Methyldopa	×
Allopurinol	

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 $\Box$ 



A 67-year-old woman presents with a rash. For the past two weeks she has felt tired and 'achey'. She also has a dry cough and some pleuritic chest pain. She is most concerned however with a new rash on her face:



Which drug is most likely to cause this presentation?



The correct answer is **Procainamide**. This patient's presentation of fatigue, dry cough, pleuritic chest pain, and a facial rash is suggestive of drug-induced lupus erythematosus (DILE). Procainamide is a well-known cause of DILE. It is an antiarrhythmic medication that can induce an autoimmune response with symptoms resembling systemic lupus erythematosus (SLE), including the characteristic malar rash seen in this patient.

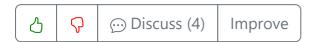
**Digoxin** is incorrect because it is a cardiac glycoside used to treat heart failure and atrial fibrillation. Its side effects include gastrointestinal disturbances, visual disturbances, and arrhythmias; however, it does not typically cause a rash or symptoms resembling lupus

erythematosus.

**Sodium valproate** is also incorrect. Sodium valproate is an anticonvulsant and mood stabilizer used in the treatment of epilepsy and bipolar disorder. Its side effects are mainly gastrointestinal, such as nausea and vomiting, as well as tremors, hair loss, and weight gain. It does not usually cause rashes or symptoms similar to lupus erythematosus.

**Methyldopa** is an alpha-2 adrenergic agonist used for the management of hypertension. Its side effects can include drowsiness, dry mouth, and headache but it does not typically cause rashes or symptoms resembling lupus erythematosus.

Finally, **Allopurinol** is incorrect. Allopurinol is a xanthine oxidase inhibitor prescribed for the treatment of gout by reducing uric acid production. While allopurinol can cause skin reactions such as a rash or Stevens-Johnson syndrome in rare cases, it does not generally lead to symptoms that resemble lupus erythematosus.



Next question >

### Drug-induced lupus \*

In drug-induced lupus not all the typical features of systemic lupus erythematosus are seen, with renal and nervous system involvement being unusual. It usually resolves on stopping the drug.

#### **Features**

- arthralgia
- myalgia
- skin (e.g. malar rash) and pulmonary involvement (e.g. pleurisy) are common
- ANA positive in 100%, dsDNA negative
- anti-histone antibodies are found in 80-90%
- anti-Ro, anti-Smith positive in around 5%



### Most common causes

- procainamide
- hydralazine

### Less common causes

- isoniazid
- minocycline
- phenytoin



Next question >



### **Textbooks**

High-yield textbook

Extended textbook

### Links

Drug-induced lupus erythematosus

Suggest link Report broken link

### Score: **14%** X 1 X 2 X 3 X 4 X 5 X 6 7 X 8 X 9 10 X 11 X 12 × 13 × 14 **V** 15 16 × × 17 × 18 × 19 20 **V** × 21 22 × × 23 × 24 25 26 27 X × 28

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Question 100 of 178





A 54-year- old lady with a back pain is being managed with 1g paracetamol four times a day and ibuprofen which has been titrated up to the maximum dose of 2.4g/daily. Although she is tolerating this well, she is still complaining of ongoing pain. What is the best option to improve her pain control?

Stop paracetamol, continue ibuprofen and commence naproxen	
Continue paracetamol, continue ibuprofen and commence naproxen	
Stop paracetamol, stop ibuprofen and commence morphine	
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Submit answer

Reference ranges  $\vee$ 

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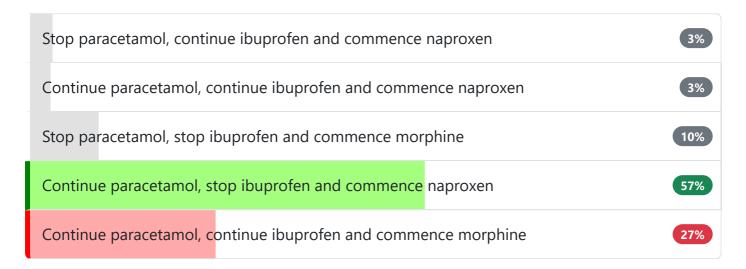
Question 100 of 178

X

 $\Box$ 

⇒

A 54-year- old lady with a back pain is being managed with 1g paracetamol four times a day and ibuprofen which has been titrated up to the maximum dose of 2.4g/daily. Although she is tolerating this well, she is still complaining of ongoing pain. What is the best option to improve her pain control?



Mild to moderate pain should be managed in a stepwise fashion:

Step 1 - paracetamol. Increase to the maximum dose of 1 gram four times a day, before switching to (or combining with) another analgesic.

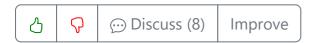
Step 2 - substitute the paracetamol with ibuprofen and increase the dose of ibuprofen to a maximum of 2.4 grams daily

Step 3 - add paracetamol to ibuprofen

Step 4 - continue with paracetamol 1 gram four times a day. Replace the ibuprofen with an alternative NSAID (such as naproxen)

Step 5 - start a full therapeutic dose of a weak opioid in addition to full-dose paracetamol (1 gram four times a day) and/or an NSAID.

Two NSAIDs should not be used concomitantly as this increases the risk of adverse effects and it is too early to start using a strong opioid such as morphine for this patient. Therefore the best answer is to continue paracetamol and switch the ibuprofen to naproxen.



Next question >

### Lower back pain \*

Lower back pain (LBP) is one of the most common presentations seen in practice. Whilst the majority of presentations will be of a non-specific muscular nature it is worth keeping in mind

possible causes which may need specific treatment.

Red flags for lower back pain

- age < 20 years or > 50 years
- history of previous malignancy
- night pain
- history of trauma
- systemically unwell e.g. weight loss, fever

The table below indicates some specific causes of LBP:

Facet joint	May be acute or chronic Pain worse in the morning and on standing On examination there may be pain over the facets. The pain is typically worse on extension of the back
Spinal stenosis	Usually gradual onset Unilateral or bilateral leg pain (with or without back pain), numbness, and weakness which is worse on walking. Resolves when sits down. Pain may be described as 'aching', 'crawling'. Relieved by sitting down, leaning forwards and crouching down Clinical examination is often normal Requires MRI to confirm diagnosis
Ankylosing spondylitis	Typically a young man who presents with lower back pain and stiffness Stiffness is usually worse in morning and improves with activity Peripheral arthritis (25%, more common if female)
Peripheral arterial disease	Pain on walking, relieved by rest  Absent or weak foot pulses and other signs of limb ischaemia  Past history may include smoking and other vascular diseases



Next question >



### **Textbooks**

Links

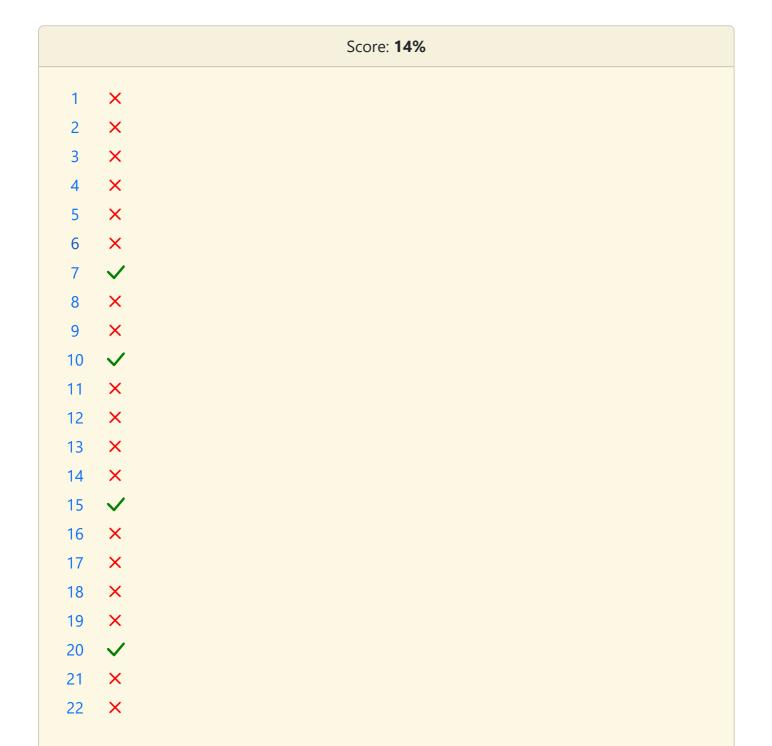
NICE

2016 Low back pain guidelines

Suggest link

Report broken link

High-yield textbook



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Question 6 of 178



 $\Box$ 



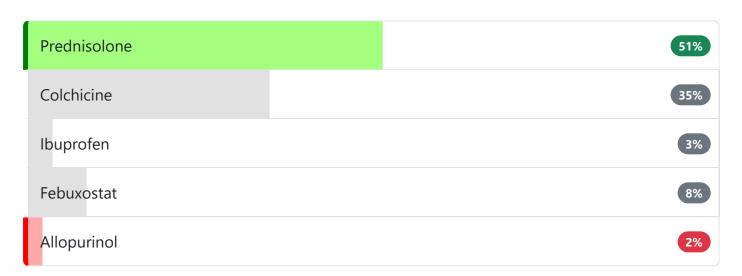
An 85-year-old man successfully completed treatment for community-acquired pneumonia. He has been medically stable for discharge but unfortunately is still an inpatient as he is awaiting a package of care to be sourced. His past medical history includes hypertension, ischaemic heart disease, type 2 diabetes and advanced chronic kidney disease (not for renal replacement therapy).

He complains of severe pain in his left foot. On examination, you note the metatarsophalangeal joint on the hallux is swollen, erythematous and hot to touch. No abnormality is detected on his right foot.

He is afebrile and scores 0 on his early warning score. His latest blood results are shown below:

Hb	120 g/L	Male: (135-180)
Platelets	454 * 10 <sup>9</sup> /L	(150 - 400)
WBC	10.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	137 mmol/L	(135 - 145)
K <sup>+</sup>	4.9 mmol/L	(3.5 - 5.0)
Urea	8.7 mmol/L	(2.0 - 7.0)
Creatinine	525 µmol/L	(55 - 120)
CRP	12 mg/L	(< 5)

Given the likely diagnosis and his co-morbidities, what would be the most appropriate treatment to commence?



For gout, if NSAIDs and colchicine are contraindicated or not tolerated the next option is a steroid



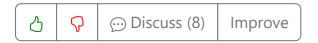
The patient is suffering from an acute attack of gout. The classical presentation is the 1st metatarsophalangeal joint being affected.

Whilst colchicine and NSAIDs such as ibuprofen are advocated by NICE as 1st line treatment for acute gout, they are contraindicated in this patient due to the severity of his CKD.

As a result, oral steroids such as prednisolone should be used.

Allopurinol is the 1st line treatment for the prevention of gout but has no benefit in an acute attack. However, if the patient is already on allopurinol, this can be continued throughout the acute flare.

Like allopurinol, febuxostat is a urate-lowering therapy used for the prevention of gout. It is used as a second-line treatment and is particularly useful in renal impairment where allopurinol is contraindicated.



Next question >

# Gout: management \*

Gout is a form of microcrystal synovitis caused by the deposition of monosodium urate monohydrate in the synovium. It is caused by chronic hyperuricaemia (uric acid >  $450 \mu mol/l$ )

#### Acute management

- NSAIDs or colchicine are first-line
  - the maximum dose of NSAID should be prescribed until 1-2 days after the symptoms have settled
  - o gastroprotection (e.g. a proton pump inhibitor) may also be indicated
- colchicine
  - inhibits microtubule polymerization by binding to tubulin, interfering with mitosis. Also inhibits neutrophil motility and activity
  - has a slower onset of action
  - may be used with caution in renal impairment: the BNF advises to reduce the dose if eGFR is 10-50 ml/min and to avoid if eGFR < 10 ml/min BNF
  - the main side-effect is diarrhoea
- oral steroids may be considered if NSAIDs and colchicine are contraindicated.
  - o a dose of prednisolone 15mg/day is usually used
- another option is intra-articular steroid injection
- if the patient is already taking allopurinol it should be continued

Indications for urate-lowering therapy (ULT)

- the British Society of Rheumatology Guidelines now advocate offering urate-lowering therapy to all patients after their **first attack of gout**
- ULT is particularly recommended if:
  - > = 2 attacks in 12 months
  - o tophi
  - o renal disease
  - uric acid renal stones
  - o prophylaxis if on cytotoxics or diuretics

#### Urate-lowering therapy

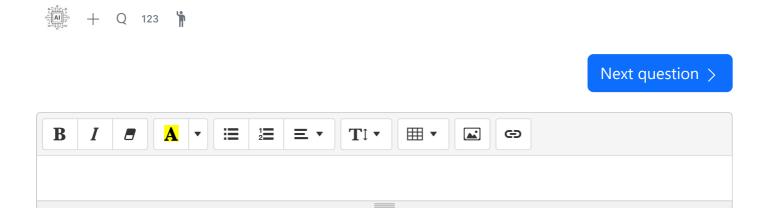
- it has traditionally been taught that urate-lowering therapy should not be started until 2 weeks after an acute attack, as starting too early may precipitate a further attack. The evidence base to support this however looks weak
- in 2017 the BSR updated their guidelines. They still support a delay in starting urate-lowering therapy because it is better for a patient to make long-term drug decisions whilst not in pain
  - the key passage is: 'Commencement of ULT is best delayed until inflammation has settled as ULT is better discussed when the patient is not in pain'
- allopurinol is first-line
  - $\circ$  initial dose of 100 mg od, with the dose titrated every few weeks to aim for a serum uric acid of < 360  $\mu$ mol/l
  - o a lower target uric acid level below 300 μmol/L may be considered for patients who have tophi, chronic gouty arthritis or continue to have ongoing frequent flares despite having a uric acid below 360 μmol/L
  - o a lower initial dose of allopurinol should be given if the patient has a reduced eGFR
  - colchicine cover should be considered when starting allopurinol. NSAIDs can be used if colchicine cannot be tolerated. The BSR guidelines suggest this may need to be continued for 6 months
- the second-line agent when allopurinol is not tolerated or ineffective is febuxostat (also a xanthine oxidase inhibitor)
- in refractory cases other agents may be tried:
  - uricase (urate oxidase) is an enzyme that catalyzes the conversion of urate to the degradation product allantoin. It is present in certain mammals but not humans
  - in patients who have persistent symptomatic and severe gout despite the adequate use of urate-lowering therapy, pegloticase (polyethylene glycol modified mammalian uricase) can achieve rapid control of hyperuricemia. It is given as an infusion once every two weeks

#### Lifestyle modifications

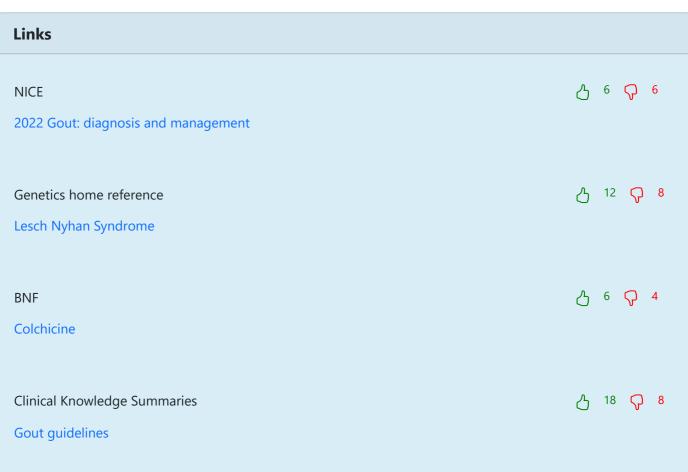
- reduce alcohol intake and avoid during an acute attack
- lose weight if obese
- avoid food high in purines e.g. Liver, kidneys, seafood, oily fish (mackerel, sardines) and yeast products

#### Other points

- consideration should be given to stopping precipitating drugs (such as thiazides)
- losartan has a specific uricosuric action and may be particularly suitable for the many patients who have coexistent hypertension
- increased vitamin C intake (either supplements or through normal diet) may also decrease serum uric acid levels







Osmosis - YouTube





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A 60-year-old man presented with worsening right hip pain and decreased mobility. He has a history of colitis and is on long-term steroids. No other medical issues were reported. He lives alone and does not smoke. He drinks two bottles of wine daily and works as a truck driver. His hip x-ray shows:



#### What is the most likely diagnosis?

Avascular necrosis of hip	
Occult fracture neck of femur	
Osteoarthritis	
Osteoporosis	
Rheumatoid arthritis	

Submit answer

Reference ranges ✓

1 -

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Question 1 of 79



 $\Box$ 



A 60-year-old man presented with worsening right hip pain and decreased mobility. He has a history of colitis and is on long-term steroids. No other medical issues were reported. He lives alone and does not smoke. He drinks two bottles of wine daily and works as a truck driver. His hip x-ray shows:



© Image used on license from Radiopaedia

What is the most likely diagnosis?



The collapse of the articular surface resulting in the crescent sign (curvilinear lucent subchondral line) is characteristic of avascular necrosis of the hip. The reduced bone density in the proximal femur is suggestive of osteopaenia.

**Avascular necrosis of hip** is the correct answer. Osteonecrosis is also called avascular necrosis (AVN) or aseptic necrosis. Although it can occur in any bone, osteonecrosis most commonly affects

the hip. It is more common in males. Risk factors include long-term steroid use, alcohol excess, and trauma.

**Occult fracture neck of femur** is incorrect. There is no evidence of disruption in the continuity of the femoral neck. The white line between the greater and lesser trochanter is the opacification from the intertrochanteric crest.

**Osteoarthritis** is incorrect. Osteoarthritis results in characteristic X-ray appearances, including joint space narrowing, osteophytes (bone spurs), articular surface cortical irregularity and sclerosis, and sub-cortical cysts (geodes) formation, which are not seen in this patient.

**Osteoporosis** is incorrect. While the proximal femur is suggestive of osteopaenia, the collapse of the articular surface resulting in the crescent sign is characteristic of avascular necrosis of the hip. In the context of the worsening hip pain, risk factors and x-ray findings, the most likely diagnosis is AVN.

**Rheumatoid arthritis** is incorrect. The radiographic hallmarks of rheumatoid arthritis are soft tissue swelling, osteoporosis, narrowing of the joint spaces and marginal erosions. Moreover, rheumatoid arthritis often affects the smaller joints first.



Next question >

# Avascular necrosis of the hip

Avascular necrosis (AVN) may be defined as death of bone tissue secondary to loss of the blood supply. This leads to bone destruction and loss of joint function. It most commonly affects the epiphysis of long bones such as the femur.

#### Causes

- long-term steroid use
- chemotherapy
- alcohol excess
- trauma

#### **Features**

- initially asymptomatic
- pain in the affected joint

#### Investigation

• plain x-ray findings may be normal initially

- o osteopenia and microfractures may be seen early on
- o collapse of the articular surface may result in the crescent sign
- MRI is the investigation of choice
  - o it is more sensitive than radionuclide bone scanning





#### Management

• joint replacement may be necessary

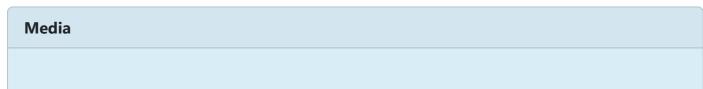


Next question >











#### Avascular necrosis of the hip

Armando Hasudungan - YouTube 0 0 0

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#### Question 2 of 79





A 54-year-old woman presents to the medical clinic with an itchy rash. She says that she has noticed a bluish-purple patchy rash mostly on sun-exposed areas. On examination, she has purple eyelids and rough raised purple areas on her knuckles Her nails show ragged cuticles, and blood vessels are seen on the nail fold. A purple, poorly defined rash is present on both her arms going up to her shoulders. What is the likely diagnosis?

Dermatomyositis	
Discoid lupus	
SLE	
Lichen planus	
Lupus pernio	

Submit answer

Reference ranges ∨

Score: **0%**1 -**2** -







Question 2 of 79



 $\Box$ 



A 54-year-old woman presents to the medical clinic with an itchy rash. She says that she has noticed a bluish-purple patchy rash mostly on sun-exposed areas. On examination, she has purple eyelids and rough raised purple areas on her knuckles Her nails show ragged cuticles, and blood vessels are seen on the nail fold. A purple, poorly defined rash is present on both her arms going up to her shoulders. What is the likely diagnosis?



The correct answer is dermatomyositis. The patient has a photosensitive rash, as well as a heliotrope rash around the eyelids, and a description of Gottron's papules. The distribution is also in keeping with a dermatomyositis rash. Lupus would normally have a macular erythematous and photosensitive butterfly rash over the face, and there can be a history of joint and neurological involvement. Lichen planus is a violaceous and itchy rash in patches, with a distribution similar to psoriasis.



Next question >

# Dermatomyositis \*

#### Overview

- an inflammatory disorder causing symmetrical, proximal muscle weakness and characteristic skin lesions
- may be idiopathic or associated with connective tissue disorders or underlying malignancy (typically ovarian, breast and lung cancer, found in 20-25% more if patient older). Screening for an underlying malignancy is usually performed following a diagnosis of dermatomyositis
- polymyositis is a variant of the disease where skin manifestations are not prominent

#### Skin features

- photosensitive
- macular rash over back and shoulder
- heliotrope rash in the periorbital region
- Gottron's papules roughened red papules over extensor surfaces of fingers
- 'mechanic's hands': extremely dry and scaly hands with linear 'cracks' on the palmar and lateral aspects of the fingers
- nail fold capillary dilatation

#### Other features

- proximal muscle weakness +/- tenderness
- Raynaud's
- respiratory muscle weakness
- interstitial lung disease: e.g. Fibrosing alveolitis or organising pneumonia
- dysphagia, dysphonia

#### Investigations

- the majority of patients (around 80%) are ANA positive
- around 30% of patients have antibodies to aminoacyl-tRNA synthetases (anti-synthetase antibodies), including:
  - o antibodies against histidine-tRNA ligase (also called Jo-1)
  - o antibodies to signal recognition particle (SRP)
  - o anti-Mi-2 antibodies

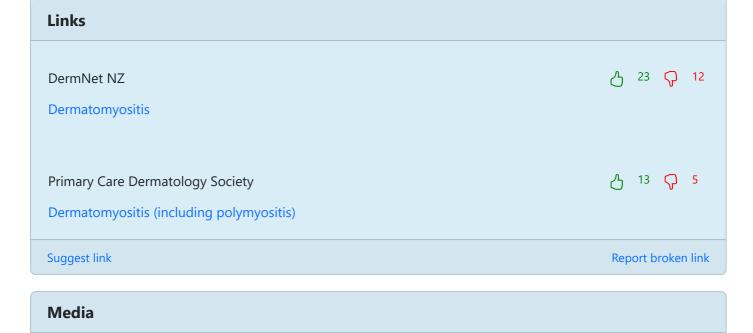


Next question >



# Textbooks High-yield textbook

Extended textbook





Polymyositis and Dermatomyositis in 3 Minutes

Townsend Teaching - YouTube









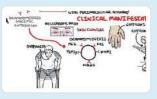
Understanding Myositis (Polymyositis and Dermatomyositis)

Zero To Finals - YouTube









**Dermatomyositis Overview** 

Armando Hasudungan - YouTube





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#### Question 3 of 79

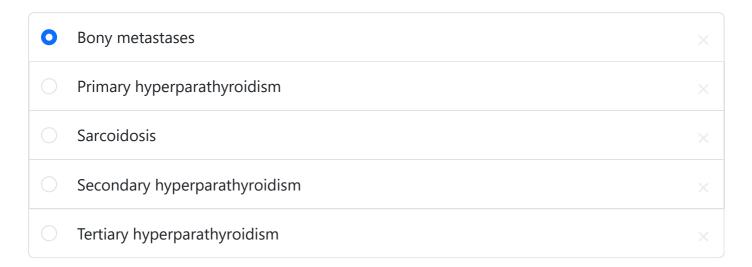




An 82-year-old woman with type 2 diabetes mellitus (T2DM), hypertension and chronic kidney disease (CKD) stage 4 is admitted under the geriatrics team with falls. Throughout her admission, her rehabilitation is significantly limited by bony pains. She is otherwise well with no systemic symptoms.

Calcium	1.82 mmol/L	(2.1-2.6)
Phosphate	2.12 mmol/L	(0.8-1.4)
ALP	# u/L	(30 - 100)
PTH	103 ng/L	(10-60)

What is the most likely explanation for her bony pains?



Submit answer

Reference ranges  $\vee$ 

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Question 3 of 79



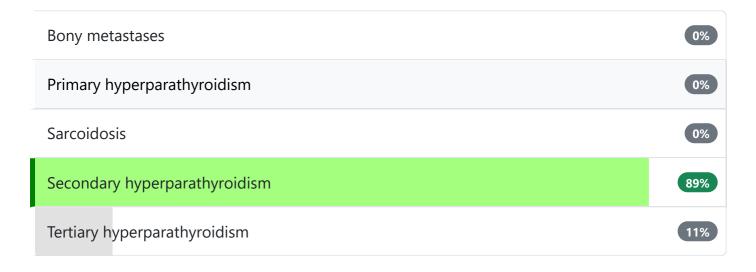
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An 82-year-old woman with type 2 diabetes mellitus (T2DM), hypertension and chronic kidney disease (CKD) stage 4 is admitted under the geriatrics team with falls. Throughout her admission, her rehabilitation is significantly limited by bony pains. She is otherwise well with no systemic symptoms.

Calcium	1.82 mmol/L	(2.1-2.6)
Phosphate	2.12 mmol/L	(0.8-1.4)
ALP	# u/L	(30 - 100)
PTH	103 ng/L	(10-60)

What is the most likely explanation for her bony pains?



Low serum calcium, raised serum phosphate, raised ALP and raised PTH - chronic kidney disease leading to secondary hyperparathyroidism

Important for me Less important

In this scenario, the patient is experiencing bony pain, and her blood tests reveal hypocalcaemia, hyperphosphataemia, elevated ALP, and raised PTH. These biochemical findings are consistent with **secondary hyperparathyroidism**, likely resulting from her CKD stage 4. In CKD, reduced phosphate excretion leads to hyperphosphataemia, and diminished vitamin D activation causes hypocalcaemia. This, in turn, triggers a physiological increase in PTH levels in an attempt to raise serum calcium. Renal mineral bone disease often presents relatively early in CKD, with bone pain being a prominent feature. Treatment focuses on normalising phosphate levels through dietary measures or phosphate binders and correcting calcium levels with calcium supplementation and activated vitamin D.

Bony metastases are an unlikely cause of this biochemical picture. While bony metastases can

increase ALP, they typically cause hypercalcaemia and a correspondingly suppressed PTH level.

In **primary hyperparathyroidism**, hypercalcaemia and an inappropriately elevated PTH are observed. Normally this is caused by a solitary parathyroid adenoma, but can also be caused by multiglandular parathyroid hypertrophy, ectopic parathyroid tissue or parathyroid cancer. Treatment options include medical management with cinacalcet, which mimics calcium to reduce PTH levels or surgical intervention with parathyroidectomy.

**Sarcoidosis** is a multisystem disease characterised by macrophage activation, with hypercalcaemia as a potential manifestation. Classic features include erythema nodosum, bilateral hilar lymphadenopathy, and hypercalcaemia. In sarcoidosis, hypercalcaemia is associated with a correspondingly suppressed PTH level, making the current biochemical picture more consistent with secondary hyperparathyroidism.

If left untreated, secondary hyperparathyroidism can progress to **tertiary hyperparathyroidism**. In this advanced state, longstanding secondary hyperparathyroidism leads to hypertrophy of the parathyroid glands, resulting in autonomous PTH production regardless of serum calcium levels. Tertiary hyperparathyroidism typically causes hypercalcaemia alongside elevated or normal PTH levels.



Next question >

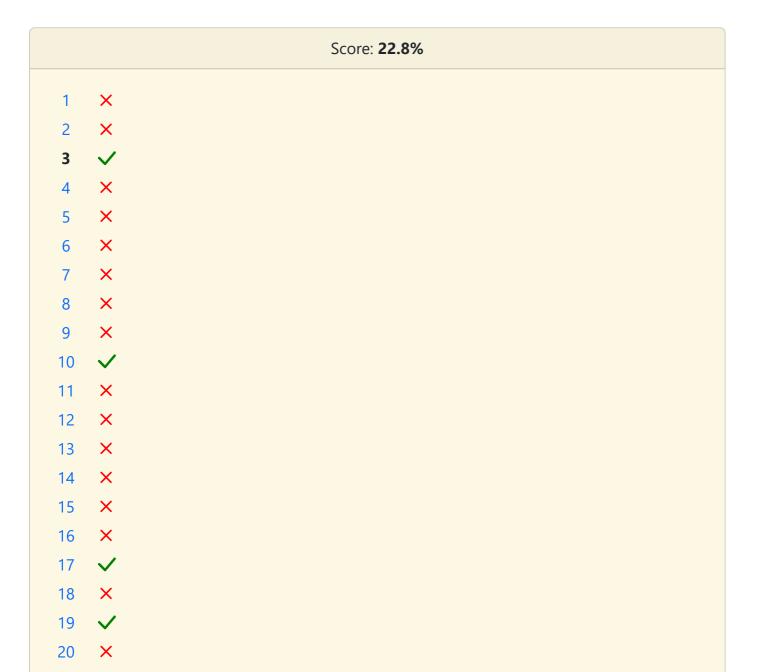
### Bone disorders: lab values \*

Disorder	Calcium	Phosphate	ALP	РТН
Osteoporosis	Normal	Normal	Normal	Normal
Osteomalacia	Decreased	Decreased	Increased	Increased
<b>Primary hyperparathyroidism</b> (→ osteitis fibrosa cystica)	Increased	Decreased	Increased	Increased
<b>Chronic kidney disease</b> (→ secondary hyperparathyroidism)	Decreased	Increased	Increased	Increased
Paget's disease	Normal	Normal	Increased	Normal
Osteopetrosis	Normal	Normal	Normal	Normal

#### **Textbooks**

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#### Question 4 of 79





A 6 year-old boy from Sierra Leone presents with a 1 week history of painful left arm. He is homozygous for sickle cell disease. On examination the child is pyrexial at 40.2°C and there is bony tenderness over the left humeral shaft. Investigations are:

Hb	7.1 g/dL
Blood culture	Gram negative rods

X-ray left humerus: Osteomyelitis - destruction of bony cortex with periosteal reaction.

What is the most likely responsible pathogen?



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Reference ranges ∨

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Question 4 of 79



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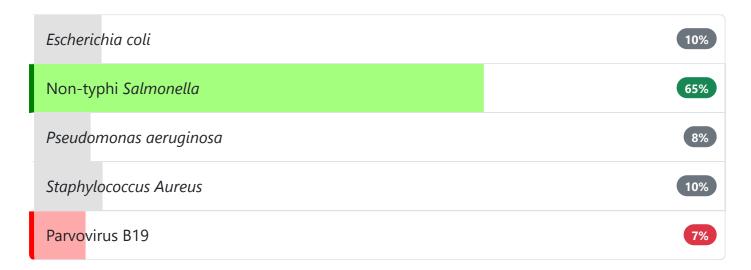


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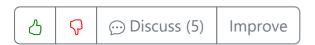
What is the most likely responsible pathogen?



Blood and bone infections caused by non-typhi salmonella (NTS) are typically associated with malaria and homozygous sickle cell disease, especially in children. The reason for this perceived susceptibility is not fully understood - but it may be in part due to the haemolysis and subsequent iron availability to the bacteria, which is 'siderophilic' in nature.

*E.coli* and *P. aeruginosa* are not typically linked to sickle cell disease and *Staphylococcus aureus* is a gram positive coccus.

The haemoglobin level is normal for a child homozygous for sickle cell disease. Therefore 'aplastic anaemia' should not be considered and parvovirus can be ruled out. Parvovirus does not cause osteomyeltitis.



# Osteomyelitis \*

Osteomyelitis describes an infection of the bone. It may be subclassified into:

- haematogenous osteomyelitis
  - o results from bacteraemia
  - o is usually monomicrobial
  - o most common form in children
  - vertebral osteomyelitis is the most common form of haematogenous osteomyelitis in adults
  - o risk factors include: sickle cell anaemia, intravenous drug user, immunosuppression due to either medication or HIV, infective endocarditis
- non-haematogenous osteomyelitis:
  - results from the contiguous spread of infection from adjacent soft tissues to the bone or from direct injury/trauma to bone
  - o is often polymicrobial
  - o most common form in adults
  - risk factors include: diabetic foot ulcers/pressure sores, diabetes mellitus, peripheral arterial disease

#### Microbiology

• *Staph. aureus* is the most common cause except in patients with sickle-cell anaemia where *Salmonella* species predominate

#### Investigations

MRI is the imaging modality of choice, with a sensitivity of 90-100%

#### Management

- flucloxacillin for 6 weeks
- clindamycin if penicillin-allergic



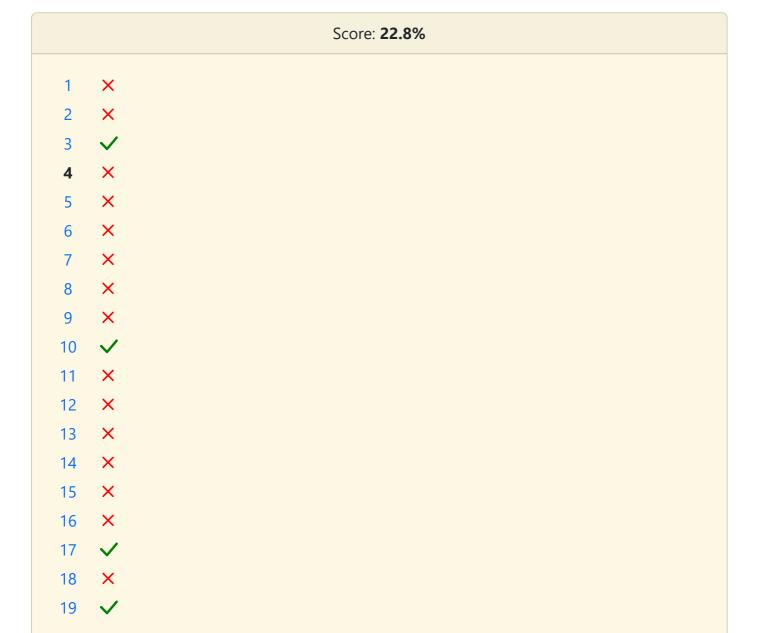
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A 77-year-old male presents to the Emergency Department with a two-day history of right temporal, throbbing headache, constant in nature and 8/10 severity. He reports this being the first ever episode of this headache and is different to his previous migraines, which have been typically in the left occipital region, lasting minutes, and fairly stereotyped over the past 60 years. Apart from migraines, he has no other medical history. On examination, his right scalp is tender and a prominent right temporal artery is noted. He is apyrexic with no skin rashes. His blood tests are as follows:

Hb	138 g/l
Platelets	552 * 10 <sup>9</sup> /l
WBC	11.5 * 10 <sup>9</sup> /l
ESR	85 mm/hr

Na <sup>+</sup>	146 mmol/l
K <sup>+</sup>	4.4 mmol/l
Urea	9.6 mmol/l
Creatinine	115 µmol/l
CRP	23 mg/l

You empirically start him on 60mg prednisolone. He undergoes temporal artery biopsy within 24 hours of his admission demonstrating no signs of temporal arteritis.

What is the most appropriate next step?

Repeat temporal artery biopsy	
Continue prednisolone but at reduced dose 10mg OD	
Discharge	
Continue prednisolone at 60mg	
Start anti-migraine medication	

Submit answer

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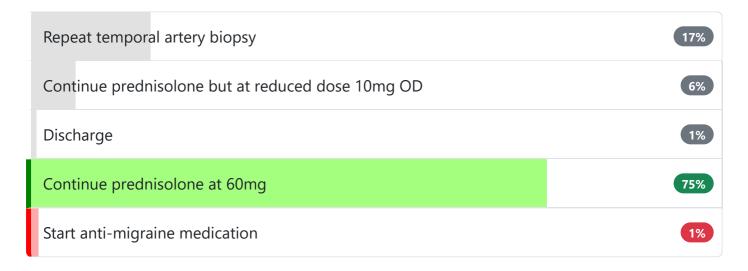
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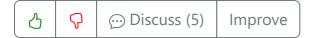
You empirically start him on 60mg prednisolone. He undergoes temporal artery biopsy within 24 hours of his admission demonstrating no signs of temporal arteritis.

What is the most appropriate next step?



All the clinical features point to right temporal arteritis. Remember that a negative temporal artery biopsy does not rule out temporal arteritis! Shorter lengths of biopsy or removal of a 'skip lesion' result in a 9% false negative rate. There is no indication for a repeat temporal artery biopsy on the

same or contralateral side, an ultrasound temporal artery may be helpful instead. Someone who has clinical temporal arteritis should thus maintain on high dose prednisolone to protect their vision. This headache is clearly different from the patient's normal migraines; anti-migraine medication is thus inappropriate.



Next question >

#### **Temporal arteritis** ★

Temporal arteritis (also known as giant cell arteritis: GCA) is a vasculitis of unknown cause that affects medium and large-sized vessels arteries. It occurs in those over 50 years old, with a peak incidence in patients who are in their 70s.

It requires early recognition and treatment to minimize the risk of complications such as permanent loss of vision. Hence, when temporal arteritis is suspected, treatment must be started promptly with high-dose prednisolone as well as urgent referral for assessment by a specialist.

There is an overlap between temporal arteritis and polymyalgia rheumatica (PMR) - around 50% of patients will have features of PMR.

#### **Features**

- typically patient > 60 years old
- usually rapid onset (e.g. < 1 month)
- headache (found in 85%)
- jaw claudication (65%)
- vision testing is a key investigation in all patients
  - anterior ischemic optic neuropathy accounts for the majority of ocular complications. It results from occlusion of the posterior ciliary artery (a branch of the ophthalmic artery) → ischaemia of the optic nerve head. Fundoscopy typically shows a swollen pale disc and blurred margins
  - o may result in temporary visual loss amaurosis fugax
  - permanent visual loss is the most feared complication of temporal arteritis and may develop suddenly
  - o diplopia may also result from the involvement of any part of the oculomotor system (e.g. cranial nerves)
- tender, palpable temporal artery
- around 50% have features of PMR: aching, morning stiffness in proximal limb muscles (not weakness)
- also lethargy, depression, low-grade fever, anorexia, night sweats

- raised inflammatory markers
  - ESR > 50 mm/hr (note ESR < 30 in 10% of patients)
  - o CRP may also be elevated
- temporal artery biopsy
  - skip lesions may be present
- note creatine kinase and EMG normal

#### **Treatment**

- urgent high-dose glucocorticoids should be given as soon as the diagnosis is suspected and before the temporal artery biopsy
  - o if there is no visual loss then high-dose prednisolone is used
  - if there is evolving visual loss IV methylprednisolone is usually given prior to starting high-dose prednisolone
  - o there should be a dramatic response, if not the diagnosis should be reconsidered
- urgent ophthalmology review
  - o patients with visual symptoms should be seen the same-day by an ophthalmologist
  - visual damage is often irreversible
- other treatments
  - bone protection with bisphosphonates is required as long, tapering course of steroids is required
  - o low-dose aspirin is sometimes given to patients as well, although the evidence base supporting this is weak

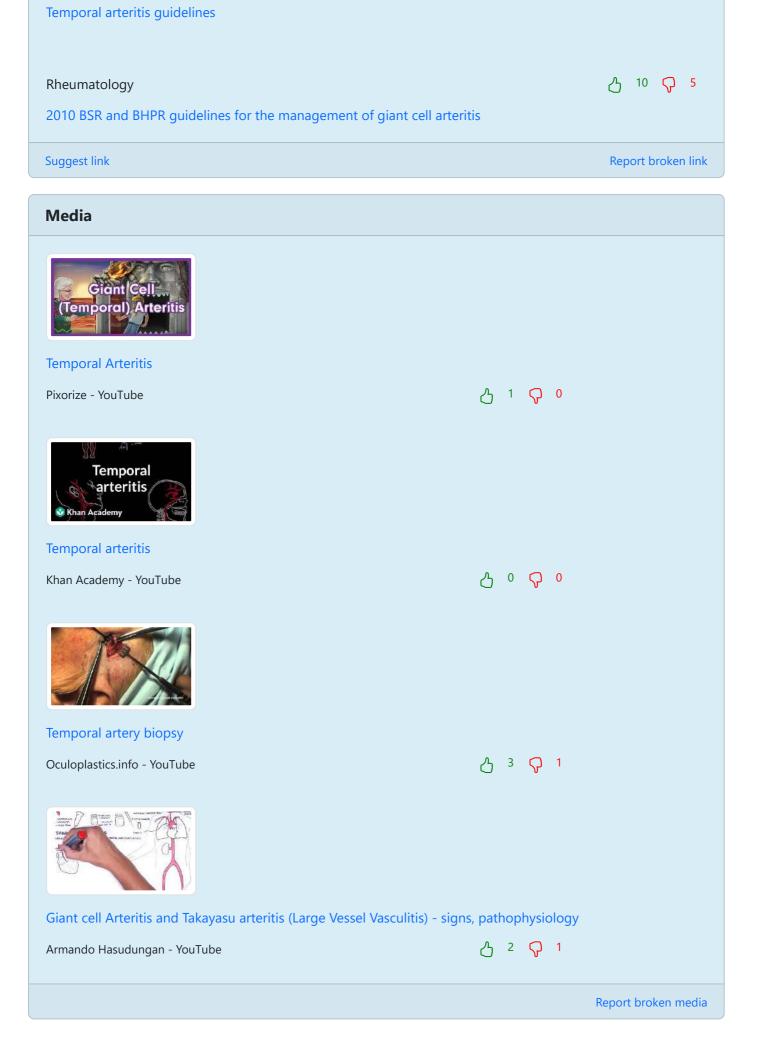


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#### Question 6 of 79

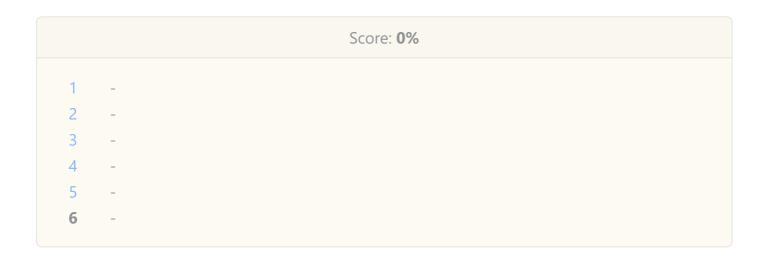
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A 32-year-old female with known rheumatoid arthritis presents to clinic and would like some advice. She would like to start a family with her partner. Her rheumatoid arthritis is current well-controlled on methotrexate and sulphasalazine, she has not required changing of doses for 2 years. She is reluctant to stop medications unless she has to, she had a number of flares when doses were reduced 3 years ago. What would you advise regarding her plans for pregnancy?

dise	She should reconsider her plans for pregnancy. Stopping medications would make her ease uncontrollable and continuing medications will affect her child	
	Continue sulphasalazine and methotrexate	
	Stop sulphasalazine, continue methotrexate	
	Continue sulphasalazine and stop methotrexate	
	Stop both sulphasalazine and methotrexate	

Submit answer

Reference ranges  $\vee$ 



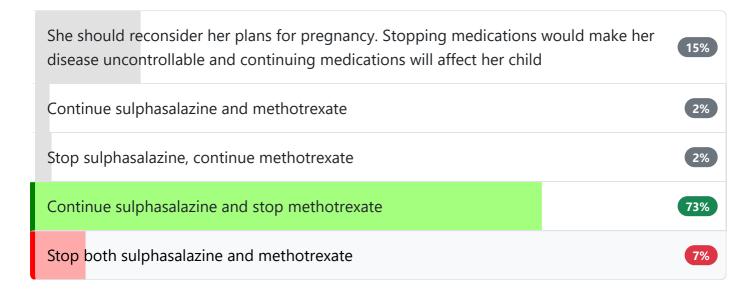




Question 6 of 79

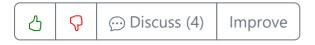
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This is a relatively common scenario in rheumatology clinics: rheumatoid arthritis has a preponderance for females and a large number of RA patients are of child-bearing age 1. The first consideration is the need for treatment, then balancing the risks of disease flares and medical foetal toxicity. The majority of RA patients, as in most autoimmune disorders, experience improvements in their condition during pregnancy. Methotrexate is highly teratogenic and should be stopped between one and three months before pregnancy. Hydroxychloroquine and sulphasalazine are generally accepted to be DMARDs that can be continued during pregnancy. Sulphasalazine should, however, be avoided in male patients attempting conception due to the risks of oligospermia. Glucocorticoids cross the placenta in low doses and can be used after 14 weeks of pregnancy. Before this cut-off, there is an increased risk of cleft palate and gestational hypertension. Low doses of prednisolone is an option should flares occur when off methotrexate.

1. Dugowson CE, Koepsell TD, Voigt LF et al. Rheumatoid arthritis in women. Incidence rates in group health cooperative, Seattle, Washington, 1987-1989. Arthritis Rheum. 1991;34(12):1502



Next question >

The management of rheumatoid arthritis (RA) has been revolutionised by the introduction of disease-modifying therapies in the past decade.

Patients with evidence of joint inflammation should start a combination of disease-modifying drugs (DMARD) as soon as possible. Other important treatment options include analgesia, physiotherapy and surgery.

#### Initial therapy

- NICE recommend DMARD **monotherapy** +/- a short-course of bridging prednisolone. In the past dual DMARD therapy was advocated as the initial step
- choices for initial DMARD monotherapy:
  - **methotrexate** is the most widely used DMARD. Monitoring of FBC & LFTs is essential due to the risk of myelosuppression and liver cirrhosis. Other important side-effects include pneumonitis
  - o sulfasalazine
  - o leflunomide
  - hydroxychloroquine: should only be considered for initial therapy if mild or palindromic disease

#### Monitoring response to treatment

 NICE recommends using a combination of CRP and disease activity (using a composite score such as DAS28) to assess response to treatment

#### Flares

flares of RA are often managed with corticosteroids - oral or intramuscular

#### **TNF-inhibitors**

- the current indication for a TNF-inhibitor is an inadequate response to at least two DMARDs including methotrexate
- etanercept: recombinant human protein, acts as a decoy receptor for TNF- $\alpha$ , subcutaneous administration, can cause demyelination, risks include reactivation of tuberculosis
- infliximab: monoclonal antibody, binds to TNF- $\alpha$  and prevents it from binding with TNF receptors, intravenous administration, risks include reactivation of tuberculosis
- adalimumab: monoclonal antibody, subcutaneous administration

#### Rituximab

- anti-CD20 monoclonal antibody, results in B-cell depletion
- two 1g intravenous infusions are given two weeks apart
- infusion reactions are common

#### Abatacept

• fusion protein that modulates a key signal required for activation of T lymphocytes

• leads to decreased T-cell proliferation and cytokine production • given as an infusion • not currently recommend by NICE + Q 123 Next question >  $\mathbf{T}$ • B ₩ • **( Textbooks** High-yield textbook Extended textbook Links △ 8 ♀ 18 NICE 2018 Rheumatoid arthritis guidelines Suggest link Report broken link Media RHEUMATOID Rheumatoid arthritis Osmosis - YouTube 💍 1 🖓 0

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#### Question 7 of 79

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A 56-year-old male presents with progressive bilateral tinnitus of gradual onset over the past 3 months. He also reports worsening hearing during the same time period, intermittent headache and increasing lower limb oedema. He denies diplopia, vertiginous symptoms, dysphagia or dysarthria. He denies any urinary symptoms or weight loss. There is a past medical history of hypertension and insulin-dependent diabetes. The patient also reports a family history of prostate carcinoma, with both his father and uncle previously undergoing resections. On examination, cranial nerves are unremarkable except bilateral hearing loss. You note no limb weakness but significant spinal kyphosis. Heart sounds I and II are heard and no added sounds. Bibasal crackles are auscultated. His abdomen is soft and non-tender. His initial serum markers are as follow:

Platelets	264 * 10 <sup>9</sup> /l
WBC	9 * 10 <sup>9</sup> /l
Neuts	5.4 * 10 <sup>9</sup> /l

Na <sup>+</sup>	142 mmol/l
K <sup>+</sup>	4.3 mmol/l
Urea	7.8 mmol/l
Creatinine	90 µmol/l

Bilirubin	6 µmol/l
ALP	902 u/l
ALT	28 u/l
CRP	16 mg/l

Parathyroid hormone and vitamin D normal range. Which other biochemical marker will be abnormal?

Corrected calcium	
Gamma glutamyltransferase (GGT)	
Phosphate	
C-telopeptide (CTx)	
Prostate specific antigen (PSA)	

#### Submit answer

Reference ranges  $\checkmark$ 

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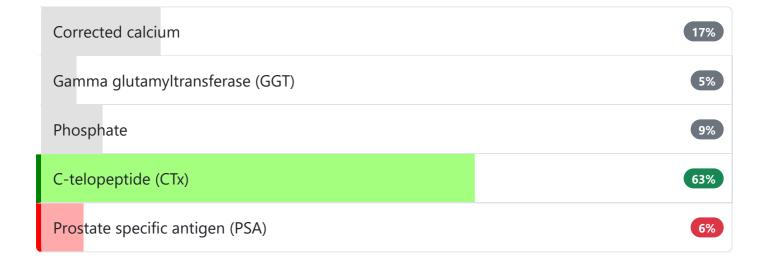
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CRP	16 mg/l

Parathyroid hormone and vitamin D normal range. Which other biochemical marker will be abnormal?



The patient describes a combination of hearing loss with tinnitus, signs of heart failure and associated skeletal deformities, associated with elevated alkaline phosphatase in the absence of other liver function test abnormalities. This is consistent with a diagnosis of Paget's disease, with skull complications (possible cochlear involvement or skull enlargement) resulting in hearing loss; high output heart failure and long bone deformities. Serum non-isomerised C-telopeptide (CTx) is an extremely sensitive marker of increased bone turnover observed in Paget's disease and is useful in monitoring disease progression or treatment efficacy<sup>1</sup>. Diagnosis is often made on clinical features, radiological imaging and raised bone-specific ALP, with exclusion of other causes of bone pathology. CTx has largely superseded old tests of urinary hydroxyproline classically used.

1. Alexandersen P, Peris P, Guanabens N, et al; Non-isomerized C-telopeptide fragments are highly sensitive markers for monitoring disease activity and treatment efficacy in Paget's disease of bone. J Bone Miner Res. 2005 Apr;20(4):588-95



Next question >

#### Paget's disease of the bone \*

Paget's disease is a disease of increased but uncontrolled bone turnover. It is thought to be primarily a disorder of osteoclasts, with excessive osteoclastic resorption followed by increased osteoblastic activity. Paget's disease is common (UK prevalence 5%) but symptomatic in only 1 in 20 patients. The skull, spine/pelvis, and long bones of the lower extremities are most commonly affected

#### Predisposing factors

- increasing age
- male sex
- northern latitude
- family history

Clinical features - only 5% of patients are symptomatic

- the stereotypical presentation is an older male with bone pain and an isolated raised ALP
- bone pain (e.g. pelvis, lumbar spine, femur)
- classical, untreated features: bowing of tibia, bossing of skull

#### Investigations

- bloods
  - raised alkaline phosphatase (ALP)

- o calcium and phosphate are typically normal. Hypercalcaemia may occasionally occur with prolonged immobilisation
- other markers of bone turnover include
  - o procollagen type I N-terminal propeptide (PINP)
  - serum C-telopeptide (CTx)
  - o urinary N-telopeptide (NTx)
  - urinary hydroxyproline
- x-rays
  - o osteolysis in early disease → mixed lytic/sclerotic lesions later
  - o skull x-ray: thickened vault, osteoporosis circumscripta
- bone scintigraphy
  - o increased uptake is seen focally at the sites of active bone lesions

#### Management

- indications for treatment include
  - o bone pain
  - skull or long bone deformity
  - o fracture
  - o periarticular Paget's
- bisphosphonate (either oral risedronate or IV zoledronate)
- calcitonin is less commonly used now

#### Complications

- deafness (cranial nerve entrapment)
- bone sarcoma (1% if affected for > 10 years)
- fractures
- skull thickening
- high-output cardiac failure





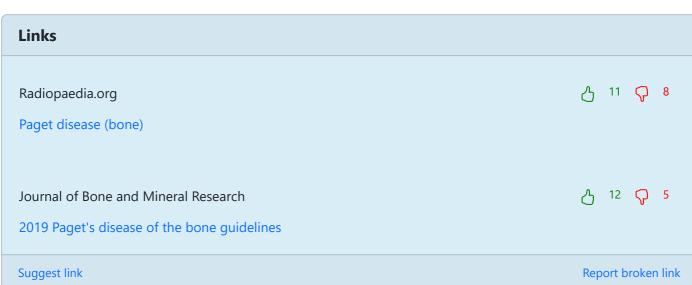




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### **Textbooks** High-yield textbook Extended textbook







#### Paget's Disease of the bone

Armando Hasudungan - YouTube  $\bigcirc$  0  $\bigcirc$  0



Paget's disease of the bone

Osmosis - YouTube





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#### Question 8 of 79

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A 20-year-old male presents with a 4-day history of joint pain in both his wrists, left 2nd metacarpal-phalangeal (MCP) joint and right knee; blood in his urine and a new rash on his cheeks, which particularly bothers him. He also complains of chest pain of non-specific nature, onset about one week ago. On examination, you note bilateral swollen MCP joints, a hyperpigmented, raised erythematous rash on both cheeks. Neurological examination reveals a mild distal tremor at rest and activity, with bilateral KayserFleischer rings. He was diagnosed with Wilsons disease aged 18 years old and has no other past medical history. He is currently a research assistant and lives alone. His medications include ibuprofen as required, penicillamine started on diagnosis 2 years ago and he states he has been buying zinc supplements over the counter after reading in a journal that it may be helpful for his condition. Urine dip demonstrates 3+ blood, 1+ protein, no leucocytes or nitrites. Which blood test is most likely to be diagnostic of his most recent admission?

Serum zinc	
Urinary zinc	
Anti-histone antibody	
Anti-C1q antibody	
Anti-double-stranded DNA antibody (dsDNA)	

Submit answer

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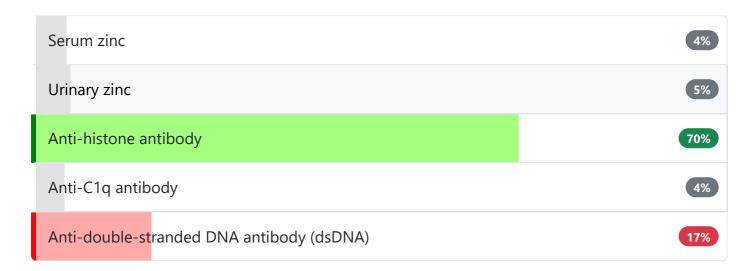
#### Question 8 of 79

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The patient describes haematuria, a new erythematous rash on sun-exposed regions and arthritis, on a background of previous penicillamine use: this is consistent with drug-induced lupus erythematosus (DLE). A number of medications are known to induce DLE, including penicillamine, procainamide, minocycline, hydralazine and a number of anti-epileptics. There is no specific diagnostic test for DLE: a combination of a drug known to induce DLE, the presence of ANA and resolution of symptoms on offending drug withdrawal. However, it is known that anti-histone antibodies are particularly sensitive to DLE, positive in up to 95% of DLE patients. Anti-C1q antibody can be present but is particularly predictive of lupus nephritis later in the disease. Unlike systemic lupus erythematosus (SLE), anti-dsDNA is often not present in DLE.

The patient's symptoms are not consistent with that of zinc poisoning, classically presenting with abdominal pain, vomiting and diarrhoea. The treatment of Wilsons disease is initially involves a copper-chelating agent, either penicillamine or trientene. Zinc is generally not used unless the patient is intolerant of either. Liver transplantation is also considered for those presenting in acute liver failure only.

#### Drug-induced lupus \*

In drug-induced lupus not all the typical features of systemic lupus erythematosus are seen, with renal and nervous system involvement being unusual. It usually resolves on stopping the drug.

#### **Features**

- arthralgia
- myalgia
- skin (e.g. malar rash) and pulmonary involvement (e.g. pleurisy) are common
- ANA positive in 100%, dsDNA negative
- anti-histone antibodies are found in 80-90%
- anti-Ro, anti-Smith positive in around 5%



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A woman with drug-induced lupus

#### Most common causes

- procainamide
- hydralazine

#### Less common causes

- isoniazid
- minocycline
- phenytoin



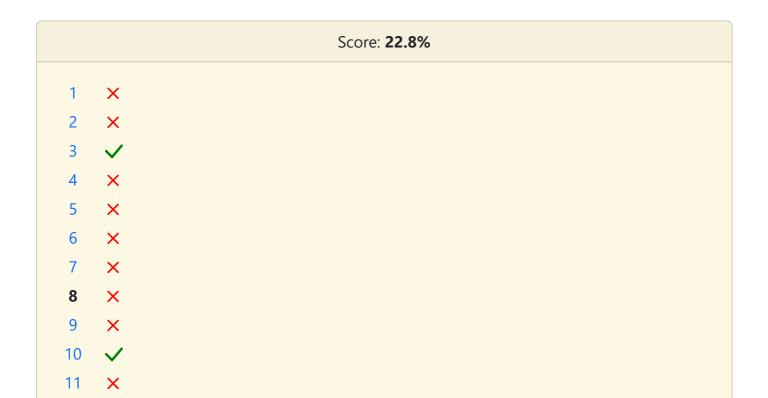
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#### **Question 9 of 79**

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A 66-year-old Caucasian female presents with 3 week history of worsening headache and 2 day history of shortness of breath. She reports disturbed sleeping at night due to an inability to lie down, due to her shortness of breath. She has no known past medical history and drug history. On examination, you note bilateral splinter haemorrhages, 4 on the right and 2 on the left, with calcium deposits distally and black spots in the pulp of the fingers. Perioral skin puckering is also noted. Cardiovascular examination is unremarkable, chest examination reveals bilateral coarse inspiratory crackles. Neurological examination is unremarkable except fundoscopy revealing papilloedema, cotton wool spots and flame haemorrhages. The patient is apyrexic, Sats 95% on 2 litres, respiratory rate 24/min, blood pressure 195/115 mmHg, HR 90/min and regular. Chest x-ray demonstrates bilateral pleural effusion with bilateral alveolar shadowing. What is the most important immediate management?

Oral amlodipine	
Oral captopril	
Intravenous labetalol	
Oral high-dose prednisolone	
Renal dialysis	

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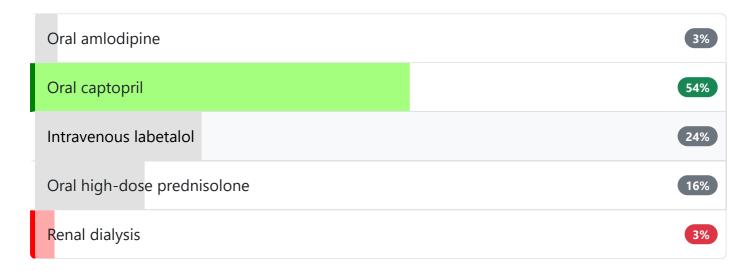
Question 9 of 79



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This patient presents with signs of cutaenous manifestations of systemic sclerosis, grade 4 hypertensive retinopathy and heart failure. She is is scleroderma renal crisis (SRC), an emergency that if left untreated, is fatal. The optimal drug of choice is an ACE inhibitor, preferably captopril, which has been trialed with the greatest experience, but other ACEi are also likely to be beneficial. In a 15 year prospective cohort, one year survival increased from 15% to 76% with the use of ACEi against other anti-hypertensives<sup>1</sup>. Steroids should be strictly avoided in SRC, they increase the risk of SRC prior to the event and may exacerbate SRC in the acute setting. Renal dialysis may be required in patients who progress to end-stage renal failure despite ACEi treatments.

1. Steen VD, Costantino JP, Shapiro AP et al. Outcome of renal crisis in systemic sclerosis: relation to availability of angiotensin converting enzyme (ACE) inhibitors. Ann Intern Med. 1990;113(5):352



# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

### Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

### Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - o patients with renal disease should be started on an ACE inhibitor
- poor prognosis

### Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis

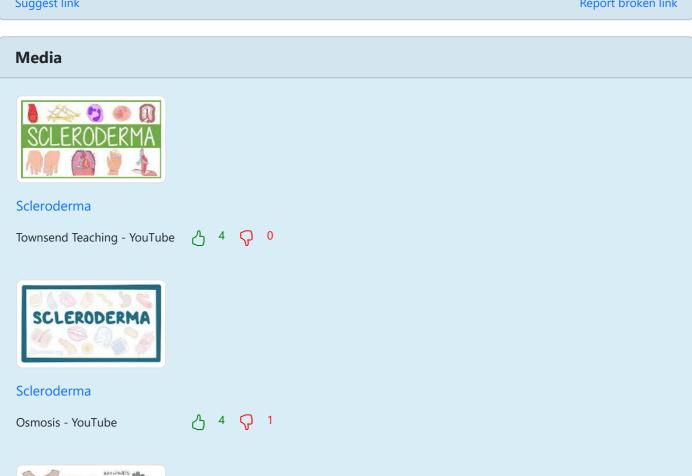


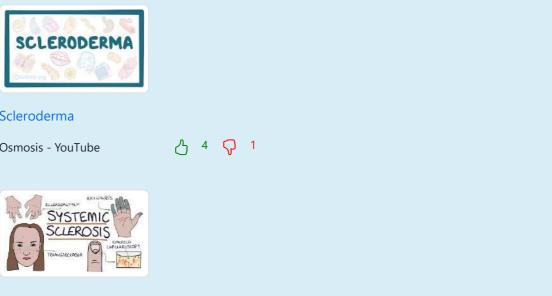












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A 70-year-old woman presents with a 2-year history of worsening hip pain on the right side. This is worse on movement but also occurs at rest, including at night. She does not have any other joints affected. Her body mass index is 32 kg/m<sup>2</sup>.

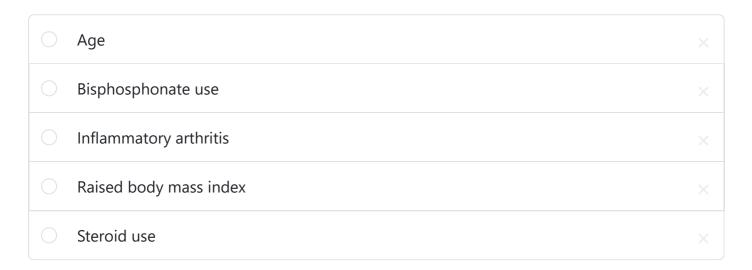
Her past medical history includes hypertension, mild asthma, and temporal arteritis.

Her pelvic x-ray is shown below.



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What is the most common risk factor associated with this condition?



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Question 10 of 79



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A 70-year-old woman presents with a 2-year history of worsening hip pain on the right side. This is worse on movement but also occurs at rest, including at night. She does not have any other joints affected. Her body mass index is 32 kg/m<sup>2</sup>.

Her past medical history includes hypertension, mild asthma, and temporal arteritis.

Her pelvic x-ray is shown below.



© Image used on license from Radiopaedia

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What is the most common risk factor associated with this condition?

Age		16%		
Bisphosphon	ate use	5%		
Inflammatory arthritis				
Raised body	mass index	15%		
Steroid use		61%		

This x-ray shows avascular necrosis of the femoral head on the right side - this is shown by the increased bone density, deformity, and flattening of the femoral head. The joint space is relatively preserved, differentiating this from osteoarthritis of the hip joint. Avascular necrosis is a type of osteonecrosis, which is due to damage to the arterial blood supply to the head of the femur. The most common cause is **steroid use**, particularly long-term steroid use. The patient in this scenario has temporal arteritis, which is often managed with prolonged high-dose steroids.

**Age** is incorrect. Increasing age is not a risk factor for AVN of the hip, outside of the presence of other risk factors. Around half of cases are present in patients less than 50 years old.

**Bisphosphonate use** is incorrect. Bisphosphonate use is associated with osteonecrosis of the jaw, but not the femoral head.

**Inflammatory arthritis** is incorrect. These conditions are not generally associated with AVN unless there is concurrent steroid use. There may be some association between systemic lupus erythematosus and AVN independent of steroid use, but this is much less common as a risk factor than steroid use in general.

**Raised body mass index** is incorrect. This is the most significant risk factor for osteoarthritis of the hip but is not a risk factor for AVN.



Next question >

# Avascular necrosis of the hip

Avascular necrosis (AVN) may be defined as death of bone tissue secondary to loss of the blood supply. This leads to bone destruction and loss of joint function. It most commonly affects the epiphysis of long bones such as the femur.

### Causes

- long-term steroid use
- chemotherapy
- alcohol excess
- trauma

### **Features**

- initially asymptomatic
- pain in the affected joint

### Investigation

- plain x-ray findings may be normal initially
  - o osteopenia and microfractures may be seen early on
  - o collapse of the articular surface may result in the crescent sign
- MRI is the investigation of choice
  - o it is more sensitive than radionuclide bone scanning





## Management

• joint replacement may be necessary

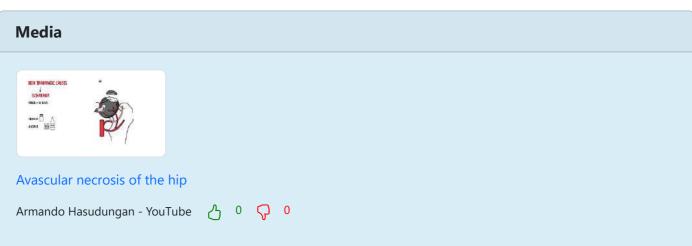


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Question 11 of 79

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A 50-year-old patient comes in with a six-month history of polyarthralgia in her hands. Blood tests show she is rheumatoid factor positive, anti-CCP antibody positive and anti nuclear antibody positive with a high titre. An ultrasound scan confirms active synovitis in the metacarpophalangeal joints of her hands bilaterally. What drug regime would you start this lady on?

Methotrexate and prednisolone	
Methotrexate and hydroxychloroquine	
Sulfasalazine and hydroxychloroquine	
Sulfasalazine, hydroxychloroquine and prednisolone	
Methotrexate, hydroxychloroquine and prednisolone	

Submit answer

Reference ranges  $\checkmark$ 

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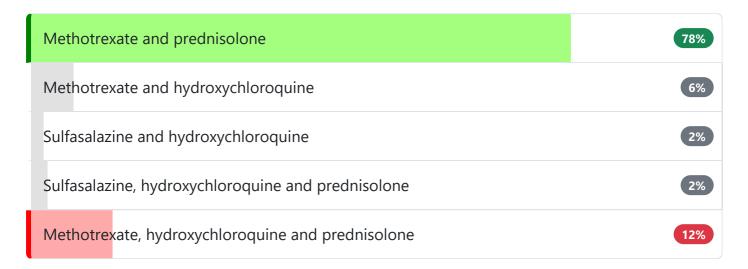


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A 50-year-old patient comes in with a six-month history of polyarthralgia in her hands. Blood tests show she is rheumatoid factor positive, anti-CCP antibody positive and anti nuclear antibody positive with a high titre. An ultrasound scan confirms active synovitis in the metacarpophalangeal joints of her hands bilaterally. What drug regime would you start this lady on?



This patient has seropositive rheumatoid arthritis. In 2018 NICE updated their guidelines. They now recommend

disease-modifying antirheumatic drug (DMARD) monotherapy with a short-course of bridging prednisolone. In the past dual DMARD therapy was advocated as the initial step.



Next question >

# Rheumatoid arthritis: management \*

The management of rheumatoid arthritis (RA) has been revolutionised by the introduction of disease-modifying therapies in the past decade.

Patients with evidence of joint inflammation should start a combination of disease-modifying drugs (DMARD) as soon as possible. Other important treatment options include analgesia, physiotherapy and surgery.

### Initial therapy

- NICE recommend DMARD monotherapy +/- a short-course of bridging prednisolone. In the past dual DMARD therapy was advocated as the initial step
- choices for initial DMARD monotherapy:

- methotrexate is the most widely used DMARD. Monitoring of FBC & LFTs is essential
  due to the risk of myelosuppression and liver cirrhosis. Other important side-effects
  include pneumonitis
- sulfasalazine
- o leflunomide
- hydroxychloroquine: should only be considered for initial therapy if mild or palindromic disease

### Monitoring response to treatment

 NICE recommends using a combination of CRP and disease activity (using a composite score such as DAS28) to assess response to treatment

### Flares

flares of RA are often managed with corticosteroids - oral or intramuscular

### **TNF-inhibitors**

- the current indication for a TNF-inhibitor is an inadequate response to at least two DMARDs including methotrexate
- etanercept: recombinant human protein, acts as a decoy receptor for TNF- $\alpha$ , subcutaneous administration, can cause demyelination, risks include reactivation of tuberculosis
- infliximab: monoclonal antibody, binds to TNF- $\alpha$  and prevents it from binding with TNF receptors, intravenous administration, risks include reactivation of tuberculosis
- adalimumab: monoclonal antibody, subcutaneous administration

### Rituximab

- anti-CD20 monoclonal antibody, results in B-cell depletion
- two 1g intravenous infusions are given two weeks apart
- infusion reactions are common

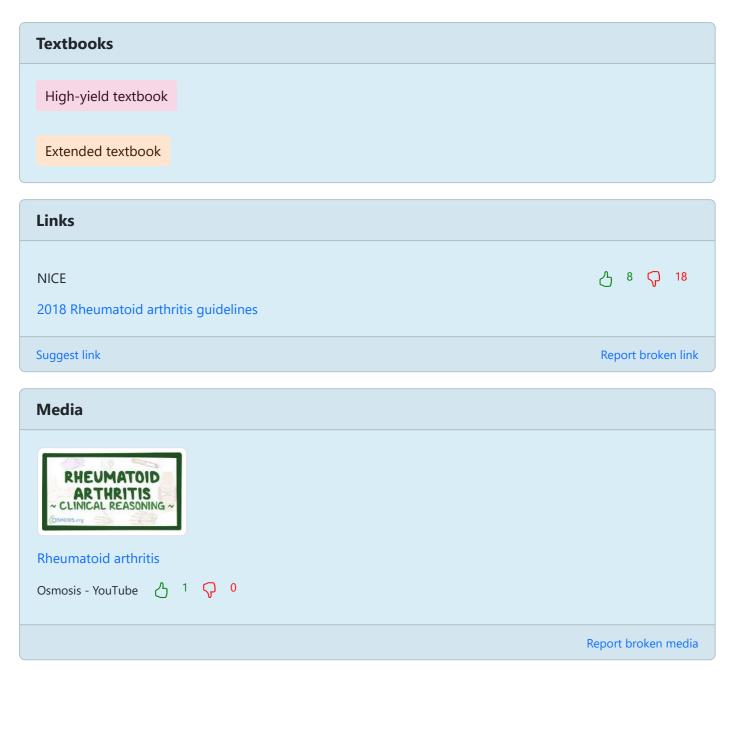
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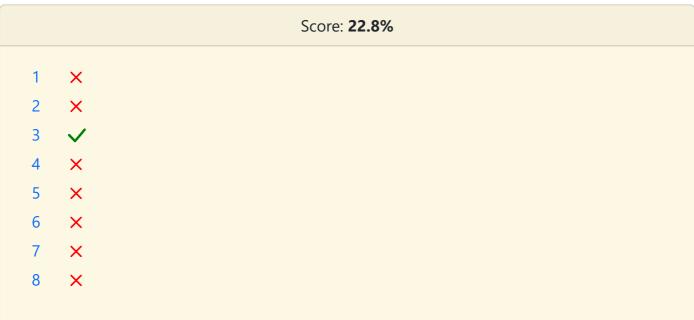
- fusion protein that modulates a key signal required for activation of T lymphocytes
- leads to decreased T-cell proliferation and cytokine production
- given as an infusion
- not currently recommend by NICE



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### Question 12 of 79





A 24-year-old who is known to have psoriasis presents with arthralgia. She has noticed that her knuckles have become swollen and her psoriasis has got much worse over the last four months. On examination, she has severe plaque psoriasis on her extensors and scalp leading to alopecia. Her metacarpophalangeal joints are clearly swollen and tender. She is currently on naproxen 500mg BD, paracetamol 1g TDS, topical steroids and calcipotriol. What medication would you add?

Leflunomide	
Sulfasalazine	
Hydroxychloroquine	
Methotrexate	
Infliximab	

### Submit answer

Reference ranges ∨

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Question 12 of 79



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Methotrexate is very effective at improving psoriatic arthritis but in addition to this it also has a dramatic effect on skin disease to a much greater extent than the other disease modifying anti-rheumatic drugs (DMARDs) listed. Therefore it is the DMARD of choice in psoriatic arthritis. If there was a contraindication to methotrexate leflunomide would be used second line for peripheral psoriatic arthritis. You would only use infliximab or another anti-TNF drugs first line if the patient had a predominantly axial spondyloarthropathy. This is according to the European League Against Rheumatism (EULAR) guidelines, please see the link: http://ard.bmj.com/content/71/1/4.full

Hydroxychloroquine can worsen skin disease and has little efficacy on psoriatic arthritis and is therefore not used routinely in the condition.



Next question >

# Psoriatic arthropathy \*

Psoriatic arthropathy is an inflammatory arthritis associated with psoriasis and is classed as one of the seronegative spondyloarthropathies. It correlates poorly with cutaneous psoriasis and often

precedes the development of skin lesions. Around 10-20% of patients with skin lesions develop an arthropathy with males and females being equally affected.

### **Presentation**

### **Patterns**

- symmetric polyarthritis
  - very similar to rheumatoid arthritis
  - o 30-40% of cases, most common type
- asymmetrical oligoarthritis: typically affects hands and feet (20-30%)
  - until recently it was thought asymmetrical oligoarthritis was the most common type,
     based on data from the original 1973 Moll and Wright paper. Please see the link for a comparison of more recent studies
- sacroiliitis
- DIP joint disease (10%)
- arthritis mutilans (severe deformity fingers/hand, 'telescoping fingers')

### Other signs

- psoriatic skin lesions
- periarticular disease tenosynovitis and soft tissue inflammation resulting in:
  - enthesitis: inflammation at the site of tendon and ligament insertion e.g. Achilles tendonitis, plantar fascitis
  - o tenosynovitis: typically of the flexor tendons of the hands
  - o dactylitis: diffuse swelling of a finger or toe
- nail changes
  - pitting
  - o onycholysis

# **Investigation and management**

### X-ray

- often have the unusual combination of coexistence of erosive changes and new bone formation
- periostitis
- 'pencil-in-cup' appearance

### Management

- should be managed by a rheumatologist
- treatment is similar to that of rheumatoid arthritis (RA). However, the following differences are noted:
  - mild peripheral arthritis/mild axial disease may be treated with 'just' an NSAID, rather than all patients being on disease-modifying therapy as with RA
  - o if more moderate/severe disease then methotrexate is typically used as in RA

- use of monoclonal antibodies such as ustekinumab (targets both IL-12 and IL-23) and secukinumab (targets IL-17)
- o apremilast: phosphodiesterase type-4 (PDE4) inhibitor → suppression of proinflammatory mediator synthesis and promotion of anti-inflammatory mediators
- o has a better prognosis than RA











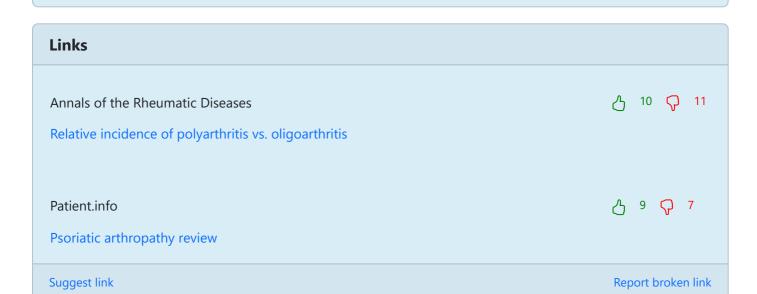
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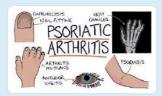
### **Textbooks**

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### Psoriatic arthritis

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### Question 13 of 79

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A 30-year-old lady with rheumatoid arthritis, presents feeling unwell. She has felt lethargic for the last two days and over the last 24 hours has developed a severe sore throat. She has been on the following prescription for the last six months; paracetamol 1g QDS, naproxen 500mg PRN, methotrexate 15mg once weekly, folic acid 5mg once weekly and prednisolone 5mg OD. She is septic on examination with observations as follows: respiratory rate 26/min, heart rate 120/min, blood pressure 100/67mmHg, temperature 37.9°C. She is tolerating oral fluids and small amounts of food. Adequate fluid resuscitation and antibiotics are started. With regards to her regular medications what should be done?

Hold methotrexate and half dose of prednisolone	
Hold methotrexate and increase prednisolone to 10mg once daily	
Half dose of methotrexate and increase prednisolone to 10mg once daily	
Refer to rheumatology for urgent review	
Hold methotrexate and start IV methylprednisolone	

Submit answer

Reference ranges  $\vee$ 

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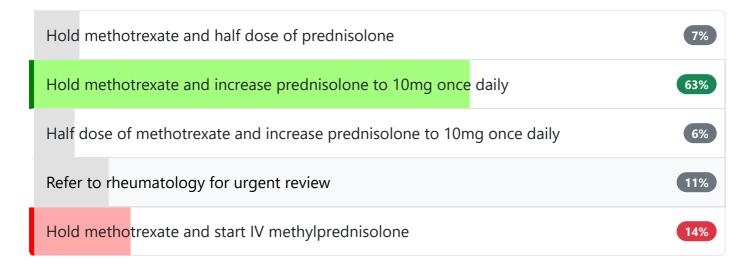
Question 13 of 79

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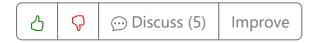
 $\Rightarrow$ 

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This lady should be presumed to be septic, she has been started on the relevant antibiotics and fluids as it states in the question. She needs her immunosuppression held and therefore no methotrexate should be given. Given that she is on long term steroids these need to be doubled to make sure she does not become Addisonian.

A referral to rheumatology would be appropriate following adequate resuscitation and treatment of the patient. Currently, her rheumatoid arthritis is not the priority.



Next question >

# Methotrexate ★

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

### Indications

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - o typically develops within a year of starting treatment, either acutely or subacutely
  - o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

### Prescribing methotrexate

- methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

### Interactions

- avoid prescribing trimethoprim or co-trimoxazole concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

### Methotrexate toxicity

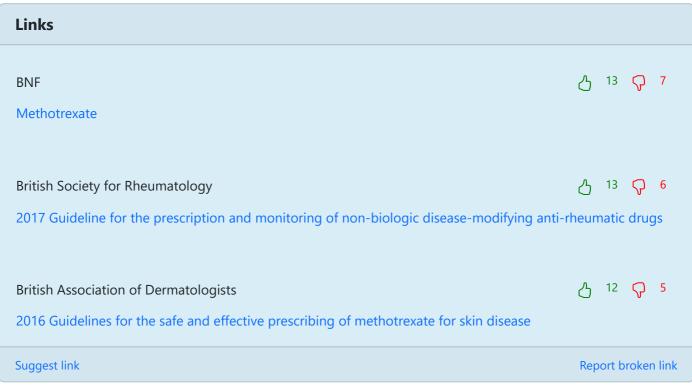
the treatment of choice is folinic acid



Next question >









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Question 14 of 79

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A 50-year-old lady presented with a 2 month history of breathlessness on exertion, increasing leg swelling and more recently a rash across her face. She was found to be in congestive heart failure, acute kidney injury and she was being treated with diuretics. Her echocardiogram showed severe left ventricular dysfunction. A few days into her admission she sustained a ventricular fibrillation cardiac arrest, which was successfully treated and the patient was started on amiodarone and betablocker. Her blood tests following the cardiac arrest did not show any electrolyte abnormalities. She also had a coronary angiogram which revealed normal coronary arteries. Subsequently, she was diagnosed with systemic lupus erythematosous and associated lupus myocarditis and she started appropriate immunosuppressive treatment for lupus.

What would be the best management for her ventricular fibrillation cardiac arrest?

	Continue amiodarone indefinitely	
	Implantable cardioverter defibrillator immediately and stop amiodarone	
dec	Continue amiodarone and medical treatment of lupus and review progress in clinic to iide about implantable cardioverter defibrillator	
	Implantable cardioverter defibrillator immediately and amiodarone indefinitely	
	None of the above	

Submit answer

Reference ranges  $\vee$ 

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Question 14 of 79

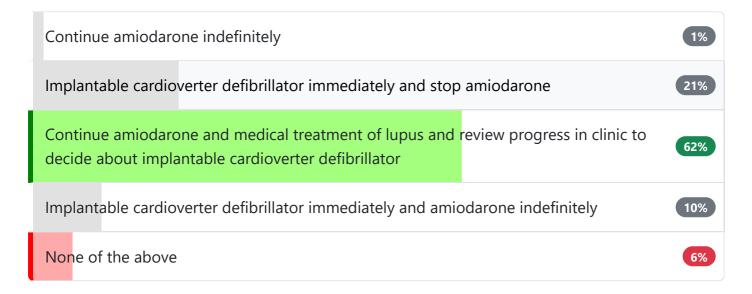


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What would be the best management for her ventricular fibrillation cardiac arrest?



This question aims to raise awareness about some of the aspects of the management of cardiac arrest survivors.

According to ACC/AHA/HRS 2008 guidelines, implantable cardioverter defibrillator (ICD) is indicated in survivors of ventricular fibrillation cardiac arrest after investigations to identify the cause of the arrest and exclude any reversible causes. Electrolyte abnormalities are among the usual reversible causes, but there is a variety of other reversible causes as well.

This patient sustained a cardiac arrest and had evidence of severe left ventricular systolic dysfunction. However, lupus myocarditis is a potentially reversible condition and so medical management is warranted before insertion of ICD. An ICD might be indicated in the near future if the patients left ventricular function does not improve following treatment of her underlying condition. This is something that can be reviewed in clinic with a repeat echocardiogram.

Amiodarone has a multitude of side effects and it should be continued indefinitely only if there is a justifiable reason to do so.

**Improve** 

Next question >

# Systemic lupus erythematosus: features \*

Systemic lupus erythematosus (SLE) is a multisystem, autoimmune disorder. It typically presents in early adulthood and is more common in women and people of Afro-Caribbean origin.

#### General features

- fatique
- fever
- mouth ulcers
- lymphadenopathy

#### Skin

- malar (butterfly) rash: spares nasolabial folds
- discoid rash: scaly, erythematous, well demarcated rash in sun-exposed areas. Lesions may progress to become pigmented and hyperkeratotic before becoming atrophic
- photosensitivity
- Raynaud's phenomenon
- livedo reticularis
- non-scarring alopecia

#### Musculoskeletal

- arthralgia
- non-erosive arthritis

#### Cardiovascular

- pericarditis: the most common cardiac manifestation
- myocarditis

#### Respiratory

- pleurisy
- fibrosing alveolitis

#### Renal

- proteinuria
- glomerulonephritis (diffuse proliferative glomerulonephritis is the most common type)

## Neuropsychiatric

- anxiety and depression
- psychosis
- seizures

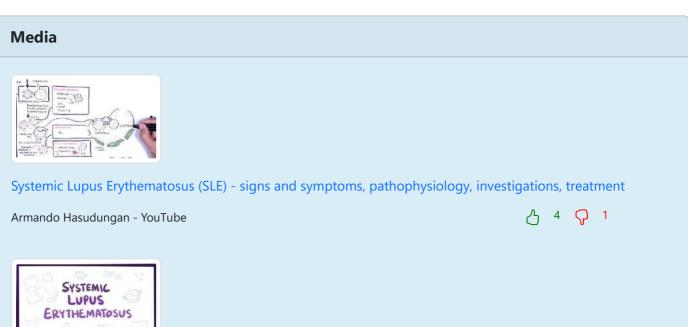


Next question >









Osmosis - YouTube

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(

A 30-year-old women with systemic lupus erythematosus is seen in the rheumatology clinic for annual follow-up. Recently she has felt well in herself and continues on hydroxychloroquine. She has not required additional steroid or analgesia for the last three years.

Her routine blood tests are as follows:

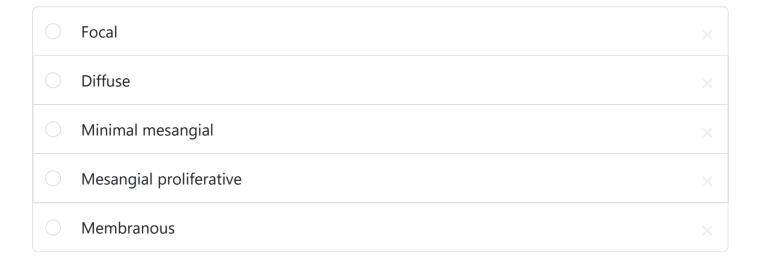
Hb	112 g/l	Na <sup>+</sup>	136 mmol/l
Platelets	252 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.9 mmol/l
WBC	6 * 10 <sup>9</sup> /l	Urea	15 mmol/l
Neuts	4.5 * 10 <sup>9</sup> /l	Creatinine	180 µmol/l
Lymphs	1 * 10 <sup>9</sup> /l	CRP	23 mg/l

Her urine dipstick shows 3+ blood and 2+ protein.

Given that her renal function was previously normal, her rheumatologist refers her for ultrasound kidneys which shows normal sized kidneys with no hydronephrosis and normal renal artery dopplers.

Following discussion at MDT, it is recommended she undergo kidney biopsy for suspected lupus nephritis.

Which class of lupus nephritis would carry the worst prognosis?



Submit answer

Reference ranges ∨

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Question 15 of 79



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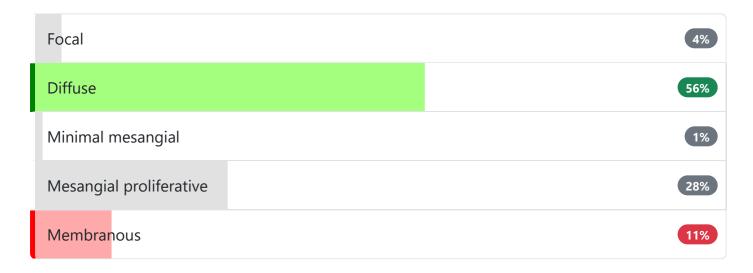
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Which class of lupus nephritis would carry the worst prognosis?



Lupus nephritis is split into 5 classes, of which class IV, diffuse proliferative, carries the worst mortality and renal outcome.

Reference: Faurschou M et al. Long term mortality and renal outcome in a cohort of 100 patients with lupus nephritis. Arthritis Care Res. 2010:62;873-880.

Next question >

# Systemic lupus erythematosus: features \*

**Improve** 

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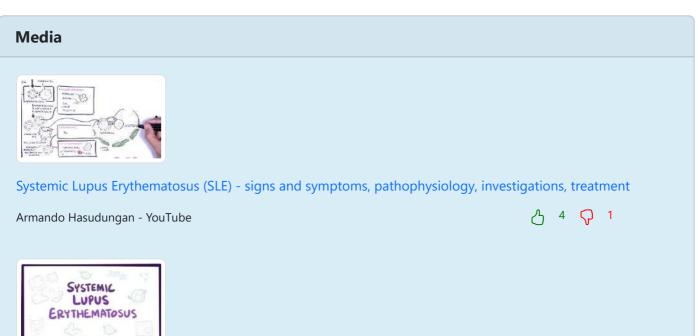


Next question >









Osmosis - YouTube

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Question 16 of 79





A 45-year-old woman presents to the rheumatology clinic with a 6-week history of painful hands and fingers. Her past medical history includes hypothyroidism for which she takes levothyroxine 125 micrograms/day and a recent diagnosis of gastro-oesophageal reflux.

On examination, her observations are within normal limits. Her hands are examined, as shown below:



What investigation will most likely confirm the probable diagnosis?

Anti-CCP antibody	
Anti-Jo antibody	
Anti-centromere antibody	
Anti-nuclear antibody	
HLA-B27 testing	

Submit answer

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Question 16 of 79

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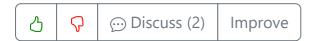
**Anti-centromere antibody** is correct. This patient's finger is erythematous with dry and tightened skin with evidence of digital ulceration. The fingernails also appear fragile and brittle with ragged cuticles. The examination findings combined with the history of painful hands, and a recent diagnosis of gastro-oesophageal reflux disease consistent with systemic sclerosis. Systemic sclerosis (or scleroderma) is an autoimmune connective tissue disease that is characterised by the fibrosis of the skin and organs secondary to the overproduction of collagen. Other symptoms include vasculopathy with Raynaud's phenomenon. Multiple antibodies can be present in systemic sclerosis. However, one of the more specific antibodies for systemic sclerosis is the anticentromere antibody that is most commonly associated with limited cutaneous systemic sclerosis.

**Anti-CCP antibody** is incorrect. Anti-CCP is associated with rheumatoid arthritis. Rheumatoid arthritis can cause painful joints, particularly smaller joints in a symmetrical distribution. Dermatological features are less commonly seen and include rheumatoid nodules and erythema nodosum. Rheumatoid arthritis does not explain this patient's presentation.

**Anti-Jo antibody** is incorrect. This is positive in dermatomyositis which is an autoimmune inflammatory disorder characterised by myopathy and dermatological manifestations including skin lesions (e.g. heliotrope rash, Gottron's papules and nail fold capillary dilatation). This is not what is seen in this patient.

**Anti-nuclear antibody** is incorrect. Although ANA antibodies are present in up to 90% of patients with systemic sclerosis, it is not a specific finding as ANA antibodies are present in numerous inflammatory conditions.

**HLA-B27 testing** is incorrect. This is present in numerous seronegative spondyloarthropathies including ankylosing spondylitis and reactive arthritis. However, neither condition gives rise to the skin changes seen in this patient.



Next question >

# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- · associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)

- other complications include renal disease and hypertension
  - o patients with renal disease should be started on an ACE inhibitor
- poor prognosis

Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







#### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis



Next question >





## Links

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### Media

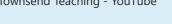


#### Scleroderma

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#### Scleroderma

Osmosis - YouTube









Systemic Sclerosis and Scleroderma

Zero to Finals - YouTube







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Question 17 of 79

P

 $\Rightarrow$ 

A 37-year-old woman with known rheumatoid arthritis was reviewed at her annual follow-up at rheumatology clinic. The diagnosis of rheumatoid arthritis had been made ten years previously after the patient experienced severe inflammation of her meta-carpal phalangeal joints of both hands. Symptoms had been controlled with an initial reducing course of oral steroids and had been subsequently maintained on 15 mg of subcutaneous methotrexate weekly. She had experience one significant flare of her symptoms 18 months previously that had necessitated a single intra-muscular dose of corticosteroids.

On this occasion, the patient reported no further swelling, pain or redness of the joints of her hands or other joints. She did report however that over the past 6 months she had experienced on-going severe pains throughout her body. In addition, she had been feeling tired and lethargic and had been finding it hard to concentrate on her work at as a computer programmer. She denied any history of skin rashes, photosensitivity, hair loss, swallowing difficulties or dry eyes and she had not lost any weight.

The examination did not demonstrate any evidence of active synovitis. A minor ulnar deviation of the digits of both hands was noted which the patient denied caused her any functional impairment. The patient was noted to be tender on palpation of the muscles of her arms, legs and paraspinal muscles. However, there was no associated muscle weakness with patient able to rise unaided from a chair without using the assistance of her arms. There was no thickening of the skin of the hands or face. The cardiovascular, respiratory and abdominal examination was unremarkable and there were no skin rashes.

Investigations requested following clinic review are listed below.

Haemoglobin	134 g / L
White cell count	6.6 * 10 <sup>9</sup> /I
Platelets	198 * 10 <sup>9</sup> /l
Sodium	139 mmol / L
Potassium	4.3 mmol / L
Urea	4.8 mmol / L
Creatinine	75 micromol / L
Erythrocyte sedimentation rate	15 mm / h
Rheumatoid factor	Positive
Anti-nuclear antigen	Negative
Anti-citrullinated protein antibodies	37 units (reference < 20)

What is the likely cause of the patient's new symptoms?

Flare of rheumatoid arthritis

Mixed connective tissue disease

Chronic regional pain syndrome

Inclusion body myositis

Fibromyalgia

Submit answer

Reference ranges  $\checkmark$ 

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Question 17 of 79







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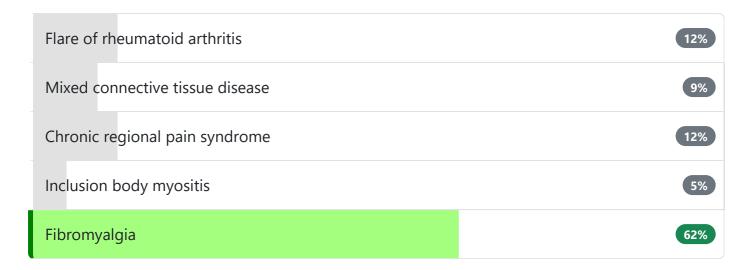
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The examination did not demonstrate any evidence of active synovitis. A minor ulnar deviation of the digits of both hands was noted which the patient denied caused her any functional impairment. The patient was noted to be tender on palpation of the muscles of her arms, legs and paraspinal muscles. However, there was no associated muscle weakness with patient able to rise unaided from a chair without using the assistance of her arms. There was no thickening of the skin of the hands or face. The cardiovascular, respiratory and abdominal examination was unremarkable and there were no skin rashes.

Investigations requested following clinic review are listed below.

Haemoglobin	134 g / L
White cell count	6.6 * 10 <sup>9</sup> /I
Platelets	198 * 10 <sup>9</sup> /l
Sodium	139 mmol / L
Potassium	4.3 mmol / L
Urea	4.8 mmol / L
Creatinine	75 micromol / L
Erythrocyte sedimentation rate	15 mm / h
Rheumatoid factor	Positive
Anti-nuclear antigen	Negative
Anti-citrullinated protein antibodies	37 units (reference < 20)

What is the likely cause of the patient's new symptoms?



The patient has chronic widespread pain associated with lethargy and difficulty concentrating and multiple tender points on palpation. The patient has immunological results consistent with her previous diagnosis of rheumatoid arthritis but no clinical or biochemical evidence of a flare of this disease or the development of a new connective tissue disease or myositis. Chronic regional pain syndrome is associated with persistent burning pain in one limb, usually after a minor injury.

The patient's symptoms are consistent with the diagnostic entity known as fibromyalgia. It is important to be aware that fibromyalgia is not a diagnosis of exclusion and can co-exist with other diseases as in this case.

Carnes D, Underwood M, Rahman A. Fibromyalgia. BMJ 2014;348:g474.



Next question >

# Fibromyalgia \*

Fibromyalgia is a syndrome characterised by widespread pain throughout the body with tender points at specific anatomical sites. The cause of fibromyalgia is unknown.

## Epidemiology

- women are around 5 times more likely to be affected
- typically presents between 30-50 years old

#### **Features**

- chronic pain: at multiple site, sometimes 'pain all over'
- lethargy
- cognitive impairment: 'fibro fog'

• sleep disturbance, headaches, dizziness are common

Diagnosis is clinical and sometimes refers to the American College of Rheumatology classification criteria which lists 9 pairs of tender points on the body. If a patient is tender in at least 11 of these 18 points it makes a diagnosis of fibromyalgia more likely

The management of fibromyalgia is often difficult and needs to be tailored to the individual patient. A psychosocial and multidisciplinary approach is helpful. Unfortunately there is currently a paucity of evidence and guidelines to guide practice. The following is partly based on consensus guidelines from the European League against Rheumatism (EULAR) published in 2007 and also a BMJ review in 2014.

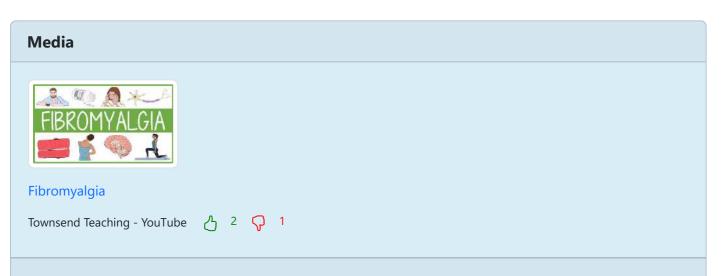
- explanation
- aerobic exercise: has the strongest evidence base
- cognitive behavioural therapy
- medication: pregabalin, duloxetine, amitriptyline



Next question >







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Question 18 of 79

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A 29-year-old woman is referred to Rheumatology clinic after experiencing all over pain throughout her body over the past two years. This has been associated with not feeling refreshed in the morning after a nights sleep and the patient finding difficulty in concentrating on her work in a call centre. She denied any history of skin rashes, photosensitivity, hair loss, swallowing difficulties or dry eyes and she had not lost any weight. Past medical history was significant for a previous diagnosis of mild depression treated with a course of cognitive behavioural therapy. There was no family history of connective tissue disease. The patient lived with her husband and two young children and reported some on-going concerns over the family finances. She smoked 10 cigarettes per day but rarely drank alcohol.

Initial review of the patient had demonstrated no evidence of inflammatory arthritis but showed the patient had significant muscular tenderness at multiple sites throughout the body.

Initial blood tests requested after clinic review had been unremarkable and included negative rheumatoid factor and negative anti-nuclear antibody. X-rays of the patient's hands and feet did not demonstrate any evidence of erosive arthropathy.

At a follow-up review of the patient with the above results, it was discussed that no evidence of inflammatory arthritis had been uncovered and that the patient's symptoms were most likely consistent with fibromyalgia. Given her previous experience with cognitive behavioural therapy, the patient was keen to adopt positive lifestyle strategies to reduce her symptoms rather than pharmacological treatment.

What treatment is most likely to be effective for this patient?

Strength training	
Balneotherapy	
Aerobic exercise	
Electrotherapy	
Acupuncture	

Submit answer

Reference ranges ✓

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Question 18 of 79







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What treatment is most likely to be effective for this patient?

Strength training	15%
Balneotherapy	3%
Aerobic exercise	70%
Electrotherapy	2%
Acupuncture	11%

Many non-pharmacological therapies for fibromyalgia have been tried although evidence for their effectiveness is limited. Aerobic exercise has the strongest evidence of benefit with a Cochrane review showing that regular aerobic exercise improved wellbeing, aerobic capacity, tenderness and pain compared with no exercise. Strength training has also been shown to have some benefit but

with a lower quality of evidence than for aerobic exercise.

There is only weak evidence to support passive physical therapies such as electrotherapy or balneotherapy (hot spa treatments). Acupuncture is often used in fibromyalgia although evidence of long-term benefit is available.

Carnes D, Underwood M, Rahman A. Fibromyalgia. BMJ 2014;348:g474.



Next question >

#### Fibromyalgia \*

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- lethargy
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- explanation
- aerobic exercise: has the strongest evidence base
- cognitive behavioural therapy
- medication: pregabalin, duloxetine, amitriptyline

Next question >



#### Textbooks

High-yield textbook

Extended textbook

#### Media



#### Fibromyalgia

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A 55-year-old man is referred to rheumatology for management of severe tophaceous gout. The patient had been experiencing intermittent gout attacks over the previous few years, typically affecting the first metatarsophalangeal joints of both feet. However, during the last two months the patient had developed inflammation of multiple small joints of his hand preventing the patient from continuing his work as a train driver. A trial of Colchicine prescribed by the patient's General Practitioner had been discontinued after the patient experienced severe diarrhoea. Past medical history included an upper GI bleed secondary to a duodenal ulcer six months previously.

Examination demonstrated severe asymmetrical inflammation of multiple metacarpalphalangeal, distal interphalangeal and proximal interphalangeal joints across both hands. Yellow-white tophi were present across the inflamed joints. Blood tests taken prior to clinic attendance are listed below.

Hb	15.2 g/dl
Platelets	265 * 10 <sup>9</sup> /l
WBC	6.5 * 10 <sup>9</sup> /l

Na <sup>+</sup>	134 mmol/l
K <sup>+</sup>	4.2 mmol/l
Urea	9.5 mmol/l
Creatinine	175 µmol/l
eGFR	62 ml/min
Urate	370 µmol/l

What is the best treatment for this patient's acute gout?

Intra-articular steroid injection	
Naproxen	
Allopurinol	
Febuxostat	
Short course prednisolone	

Submit answer

Reference ranges  $\vee$ 

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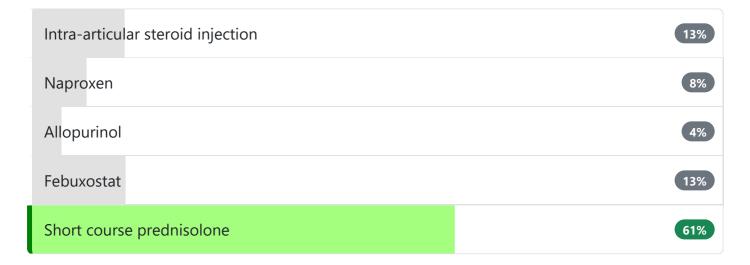
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Creatinine	175 µmol/l
eGFR	62 ml/min
Urate	370 µmol/l

What is the best treatment for this patient's acute gout?



The best option in this case is a short course of oral prednisolone (30 mg daily for five days). Two

randomised controlled trials have shown this treatment to have a similar efficacy compared to NSAIDs. Intra-articular steroid injection is felt to be an effective treatment for acute gout affecting large joints, but is less appropriate for treatment of multiple small joints of the hands. Naproxen would be contra-indicated in this case due to the history of peptic ulceration and renal impairment.

Allopurinol and Febuxostat are both used to lower serum urate levels as part of gout prophylaxis and have no role in the treatment of an acute attack.

Roddy E, Mallen C, Doherty M. Gout. BMJ 2013;347:5648.



Next question >

#### Gout: management \*

Gout is a form of microcrystal synovitis caused by the deposition of monosodium urate monohydrate in the synovium. It is caused by chronic hyperuricaemia (uric acid >  $450 \mu mol/l$ )

#### Acute management

- NSAIDs or colchicine are first-line
  - the maximum dose of NSAID should be prescribed until 1-2 days after the symptoms have settled
  - o gastroprotection (e.g. a proton pump inhibitor) may also be indicated
- colchicine
  - inhibits microtubule polymerization by binding to tubulin, interfering with mitosis. Also inhibits neutrophil motility and activity
  - has a slower onset of action
  - may be used with caution in renal impairment: the BNF advises to reduce the dose if eGFR is 10-50 ml/min and to avoid if eGFR < 10 ml/min (BNF)
  - o the main side-effect is diarrhoea
- oral steroids may be considered if NSAIDs and colchicine are contraindicated.
  - o a dose of prednisolone 15mg/day is usually used
- another option is intra-articular steroid injection
- if the patient is already taking allopurinol it should be continued

#### Indications for urate-lowering therapy (ULT)

- the British Society of Rheumatology Guidelines now advocate offering urate-lowering therapy to all patients after their first attack of gout
- ULT is particularly recommended if:
  - > = 2 attacks in 12 months

- o tophi
- o renal disease
- uric acid renal stones
- o prophylaxis if on cytotoxics or diuretics

#### **Urate-lowering therapy**

- it has traditionally been taught that urate-lowering therapy should not be started until 2 weeks after an acute attack, as starting too early may precipitate a further attack. The evidence base to support this however looks weak
- in 2017 the BSR updated their guidelines. They still support a delay in starting urate-lowering therapy because it is better for a patient to make long-term drug decisions whilst not in pain
  - the key passage is: 'Commencement of ULT is best delayed until inflammation has settled as ULT is better discussed when the patient is not in pain'
- allopurinol is first-line
  - $\circ$  initial dose of 100 mg od, with the dose titrated every few weeks to aim for a serum uric acid of < 360  $\mu$ mol/l
  - o a lower target uric acid level below 300 μmol/L may be considered for patients who have tophi, chronic gouty arthritis or continue to have ongoing frequent flares despite having a uric acid below 360 μmol/L
  - o a lower initial dose of allopurinol should be given if the patient has a reduced eGFR
  - colchicine cover should be considered when starting allopurinol. NSAIDs can be used if colchicine cannot be tolerated. The BSR guidelines suggest this may need to be continued for 6 months
- the second-line agent when allopurinol is not tolerated or ineffective is febuxostat (also a xanthine oxidase inhibitor)
- in refractory cases other agents may be tried:
  - uricase (urate oxidase) is an enzyme that catalyzes the conversion of urate to the degradation product allantoin. It is present in certain mammals but not humans
  - in patients who have persistent symptomatic and severe gout despite the adequate use of urate-lowering therapy, pegloticase (polyethylene glycol modified mammalian uricase) can achieve rapid control of hyperuricemia. It is given as an infusion once every two weeks

#### Lifestyle modifications

- reduce alcohol intake and avoid during an acute attack
- lose weight if obese
- avoid food high in purines e.g. Liver, kidneys, seafood, oily fish (mackerel, sardines) and yeast products

#### Other points

- consideration should be given to stopping precipitating drugs (such as thiazides)
- losartan has a specific uricosuric action and may be particularly suitable for the many patients who have coexistent hypertension

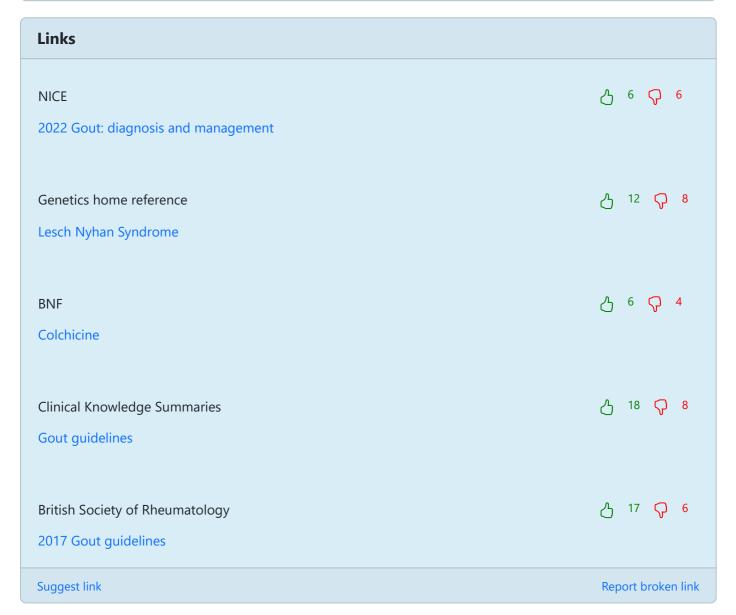
• increased vitamin C intake (either supplements or through normal diet) may also decrease serum uric acid levels



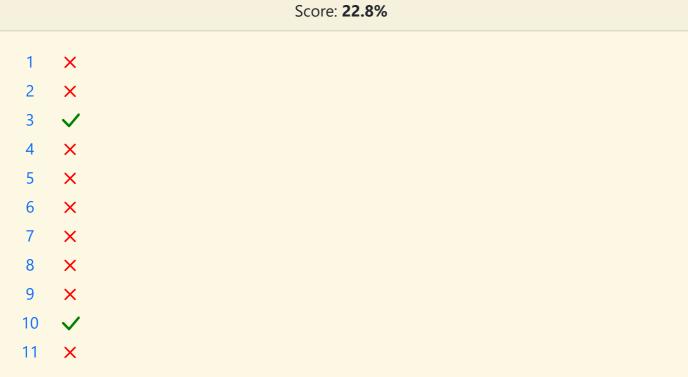
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Question 20 of 79

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A 78-year-old female falls whilst taking out the bins. She immediately has severe right hip pain and her right leg is shortened and externally rotated. She is taken to the Emergency Department where an X-ray is performed, which confirms a right-sided intracapsular neck of femur fracture. She has definite operative management the following day.

Following surgery, how should this patient's bone health be managed to reduce the risk of further fragility fractures?

DEXA scan before prescribing	
MRI scan of the spine and pelvis before prescribing	
Start alendronate, no imaging required	
Start calcium and vitamin D, no imaging required	
Start calcium and vitamin D whilst awaiting a DEXA scan	

Submit answer

Reference ranges ∨

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Question 20 of 79

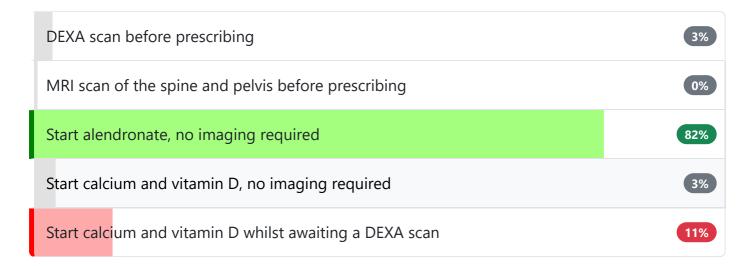


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A 78-year-old female falls whilst taking out the bins. She immediately has severe right hip pain and her right leg is shortened and externally rotated. She is taken to the Emergency Department where an X-ray is performed, which confirms a right-sided intracapsular neck of femur fracture. She has definite operative management the following day.

Following surgery, how should this patient's bone health be managed to reduce the risk of further fragility fractures?



Start alendronate in patients >= 75 years following a fragility fracture, without waiting for a DEXA scan

Important for me Less important

In patients over the age of 75 who present with a fragility fracture a diagnosis of osteoporosis is presumed and does not need to be confirmed with imaging. Starting first-line therapy without a DEXA scan is recommended in this age group. First-line therapy is currently alendronate 70 mg once per week.

In patients under 75 years who present with a fragility fracture, a DEXA scan is recommended prior to prescribing alendronate. With the results of the DEXA scan, a FRAX assessment is completed to assess the patient's future fracture risk and provides guidance on prescribing on this basis. The patient in this question is over 75-years-old.

The preferred method of imaging to assess the severity of osteoporosis is a DEXA scan rather than an MRI scan. An MRI scan may be useful in the context of a pathological malignancy when underlying malignancy is suspected.

Vitamin D and calcium replacement is not an adequate treatment for osteoporosis to prevent further fragility fractures.

As above, vitamin D and calcium replacement is not an adequate treatment for osteoporosis to prevent further fragility fractures. Furthermore, this patient is over 75-years-old and therefore does not require a DEXA scan to make the diagnosis of osteoporosis.



Next question >

#### Osteoporosis: Assessing patients following a fragility fracture



The management of patients following a fragility fracture depends on age.

#### Patients >= 75 years of age

Patients who've had a fragility fracture and are >= 75 years of age are presumed to have underlying osteoporosis and should be started on first-line therapy (an oral bisphosphonate), without the need for a DEXA scan.

It should be noted that the 2014 NOGG guidelines have a different threshold, suggesting treatment is started in all women over the age of 50 years who've had a fragility fracture -'although BMD measurement may sometimes be appropriate, particularly in younger postmenopausal women.'

#### Patients < 75 years of age

If a patient is under the age of 75 years a DEXA scan should be arranged. These results can then be entered into a FRAX assessment (along with the fact that they've had a fracture) to determine the patients ongoing fracture risk.

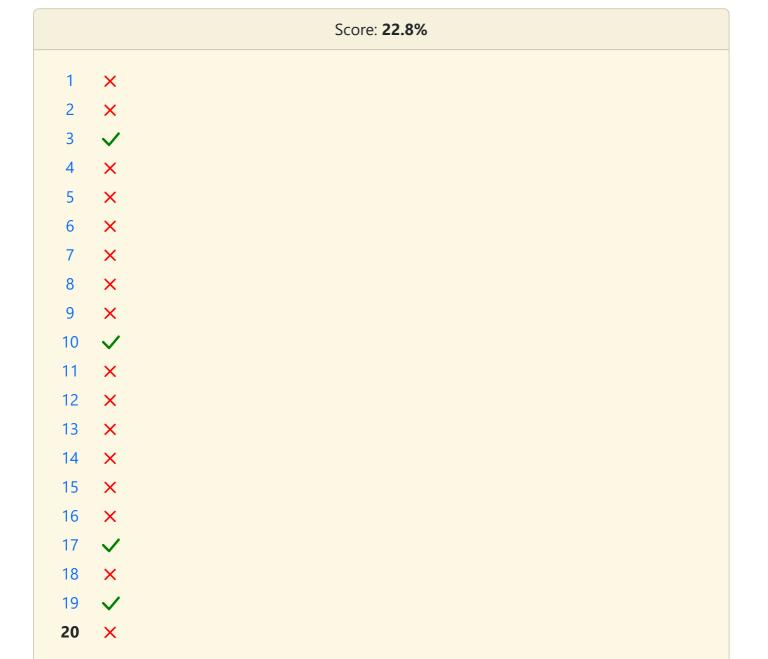
For example, a 79-year-old woman falls over on to an outstretched hand and sustains a Colles' fracture (fracture of the distal radius). Given her age she is presumed to have osteoporosis and therefore started on oral alendronate 70mg once weekly. No DEXA scan is arranged.



Next question >



# Textbooks High-yield textbook Extended textbook Links The National Osteoporosis Guideline Group NOGG Pocket Guide for Healthcare Professionals Suggest link Report broken link



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Question 21 of 79

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A 64-year-old male presents with known lumbosacral poly-radiculopathy caused by neurosarcoidosis is on immunosuppression with high dose steroids for several months and methotrexate. He is admitted with breathlessness and a dry cough. He is found to have a type 1 respiratory failure. A CT chest shows ground glass shadowing suggesting *Pneumocystis jirovecii* pneumonia. You discuss with the microbiology team who suggest also covering the patient for a bacterial pneumonia and viral pneumonitis.

Which of the following medications could have a potentially life threatening interaction with his current medications?

Tazocin	
Clarithromycin	
Co-trimoxazole	
Ciprofloxacin	
Aciclovir	

Submit answer

Reference ranges  $\vee$ 

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Question 21 of 79



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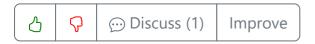


Co-trimoxazole contains trimethoprim and therefore should never be prescribed with methotrexate

Important for me Less important

Co-trimoxazole is a combination of sulfamethoxazole and trimethoprim. These are bacteriostatic antibiotics that work by interfering with bacterial folate metabolism. However, they can also affect the hosts folate metabolism. Methotrexate is a dihydrofolate reductase inhibitor. Therefore, together these medications can cause potentially life-threatening bone marrow suppression secondary to folate deficiency.

The BNF does not list interactions between corticosteroids and the remaining medication listed. Ciprofloxacin may possibly reduce methotrexate excretion, so monitoring for signs of potential toxicity needs to be undertaken. No interactions are listed in the BNF between methotrexate, and tazocin, aciclovir and clarithromycin.



#### Methotrexate ★

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

#### Indications

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

#### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - o typically develops within a year of starting treatment, either acutely or subacutely
  - o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

#### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

#### Prescribing methotrexate

- methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

#### Interactions

- avoid prescribing trimethoprim or co-trimoxazole concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

#### Methotrexate toxicity

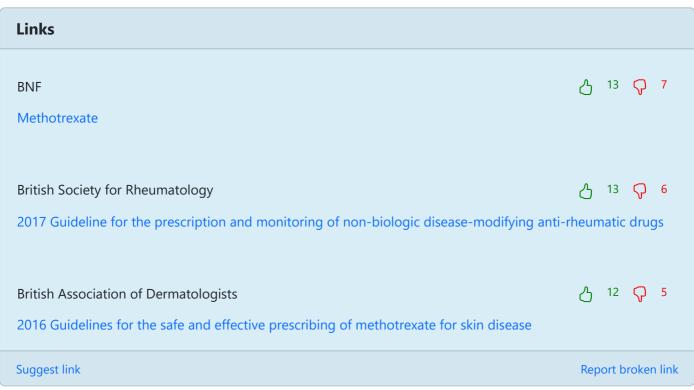
• the treatment of choice is folinic acid



Next question >







#### Media



Methotrexate - Pharmacology (DMARDs, mechanism of action, side effects)

Armando Hasudungan - YouTube





Report broken media

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Back to top







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A 38-year-old Armenian visitor presents with 3 day history of pyrexia, shortness of breath, chest pain and abdominal pain, associated with temperature of 38.5 degrees. She has no other known past medical history and reports at least 2 other episodes of similar pain, both times spontaneously resolving without treatment or diagnosis. On examination, she has a pleural rub and a swollen, tender left 3rd metcarpal-phalangeal joint. Her mother has recently been admitted for similar symptoms last month. Her blood tests are as follow:

Hb	14.5 g/dl
Platelets	560 * 10 <sup>9</sup> /l
WBC	17.8 * 10 <sup>9</sup> /l

Na <sup>+</sup>	143 mmol/l
K <sup>+</sup>	4.6 mmol/l
Urea	5.2 mmol/l
Creatinine	78 µmol/l
CRP	78 mg/l

A chest radiograph demonstrates mild bilateral pleural effusions with no significant focus of consolidation, her Mantoux test is negative. Urine dip is negative, urine MC+S grows no organisms, urinary porphobilinogen is negative. A rheumatology review was requested regarding the synovitis and colchicine prescribed. She responds well with resolution of all symptoms within 24 hours. An infectious diseases opinion and induced sputum is awaited. What is the most likely diagnosis?

Tuberculosis	
Acute intermittent porphyria (AIP)	
Coxsackie B virus infection	
Familial mediterranean fever	
Systemic lupus erythematous (SLE)	

Submit answer

Reference ranges  $\vee$ 

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Question 22 of 79





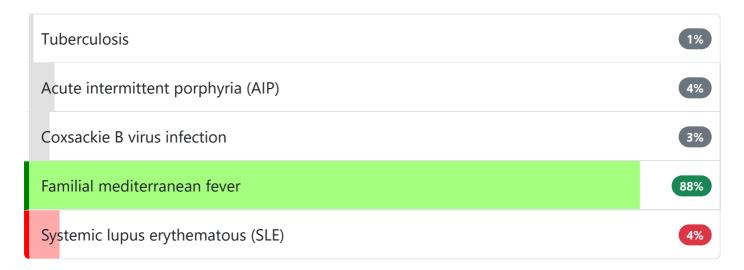


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Familial Mediterranean fever (FMF) is an autosomal recessive, hereditary inflammatory disorder characterised by reoccurring episodes of abdominal pain, fever, arthralgia, and chest

The patient is of Mediterranean descent is experiencing an acute attack of abdominal pain, chest pain, synovitis and pyrexia with an acute phase response. There appears to be a previous history of similar symptoms and a possible genetic element. Familial Mediterranean fever would fit with all these symptoms, with almost all patients presenting with abdominal pain, pleuritis and synovitis, associated with fever greater than 38 degrees. The main differentials in this case are with SLE and AIP: during an acute event, urinary porphobilinogen is likely positive in AIP. The distinguishing feature against SLE is the resolution of symptoms with colchicine, which is a key diagnostic feature<sup>1</sup>.

1. Livneh A, Langevitz P, Zemer D et al. Criteria for the diagnosis of familial Mediterranean fever. Arthritis Rheum. 1997;40(10):1879



Next question >

# Familial Mediterranean Fever \*

Familial Mediterranean Fever (FMF, also known as recurrent polyserositis) is an autosomal recessive disorder which typically presents by the second decade. It is more common in people of Turkish, Armenian and Arabic descent.

Features - attacks typically last 1-3 days

- pyrexia
- abdominal pain (due to peritonitis)
- pleurisy
- pericarditis
- arthritis
- erysipeloid rash on lower limbs

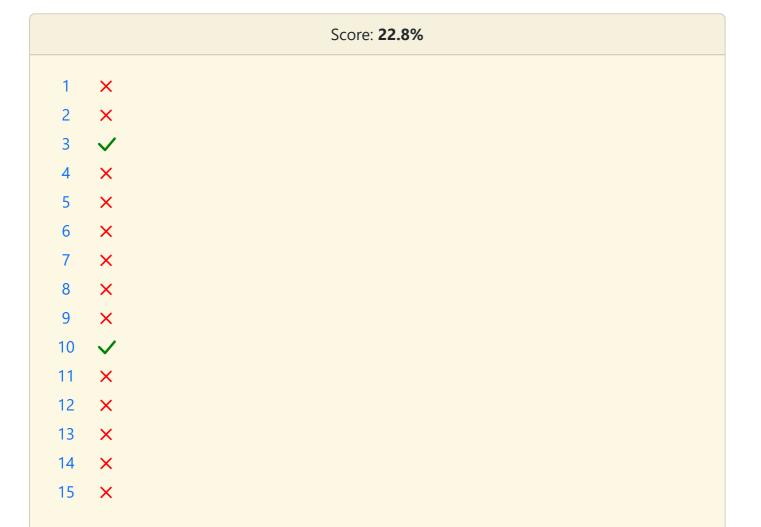
### Management

colchicine may help









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#### Question 23 of 79





A 65-year-old gentleman is reviewed in rheumatology clinic, as he has been referred by his general practitioner regarding his recurrent gout. The patient had already been started on allopurinol due to recurrent attacks of gouts. Unfortunately he was unable to tolerate it, as he developed a rash. In addition, he was not happy with the gastrointestinal upset that it caused him. Furthermore, his eGFR (see bloods) does not permit an increase of allopurinol above 100 mg OD.

On examination today you note an obese gentleman who is otherwise asymptomatic. He informs you that in the past the gout attacks affected his 1st metatarsophalangeal joint in the left foot and his right knee.

He is known to suffer from hypertension, type II diabetes mellitus and has had previous myocardial infarction. He is on aspirin 75 mg OD, ramipril 3.25 mg OD, amlodipine 5 mg OD, atorvastatin 80 mg OD, bisoprolol 1.25 mg OD, metformin MR 1 g OD.

You review his bloods and his previous aspiration results.

## 3 months prior

Na <sup>+</sup>	137 mmol/l
K <sup>+</sup>	4.3 mmol/l
Urea	6.8 mmol/l
Creatinine	123 µmol/l

#### Now

Na <sup>+</sup>	138 mmol/l
K <sup>+</sup>	5.2 mmol/l
Urea	10.3 mmol/l
Creatinine	235 µmol/l

What options exist with regards to his long-term management of gout?

Continue with allopurinol 100 mg OD	
Initiate febuxostat 80 mg OD	
Initiate indomethacin MR 75 mg OD	
Initiate prednisolone 30 mg OD	

# Submit answer

Reference ranges ✓

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Question 23 of 79



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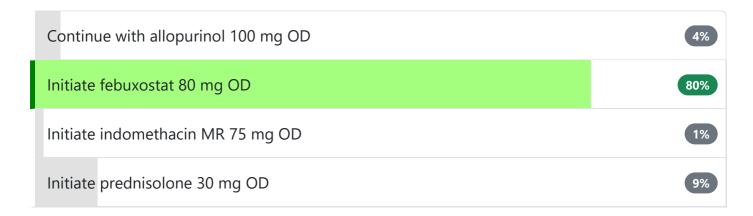
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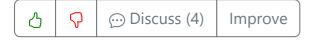
What options exist with regards to his long-term management of gout?



Febuxostat can be used in the prevention of gout in patients where the allopurinol side effects have been deemed intolerable or the renal function does not allow sufficient dose escalation

Important for me Less important

NICE guidelines alongside the British Society of Rheumatology guidelines on the management of gout suggest that febuxostat is an alternative to allopurinol in cases where the side-effects are deemed bad by the patient and no effective dose adjustment to 300 mg OD has been made due to declining renal function. Febuxostat is a non-purine selective xanthine oxidase inhibitor, inhibiting oxidised and reduced versions of the enzyme. It does not inhibit purine or pyrimidine metabolism as allopurinol does. Recent studies do mention that there may be an elevated risk of cardiovascular events with the use of febuxostat, however no change in guidelines has been made. Indomethacin (an NSAID), prednisolone and colchicine are all useful in the acute treatment of gout, however their preventative use is limited. Colchicine can be used as cross cover while a xanthine oxidase inhibitor is first prescribed to avoid acute gout.



Next question >

# Gout: management \*

Gout is a form of microcrystal synovitis caused by the deposition of monosodium urate monohydrate in the synovium. It is caused by chronic hyperuricaemia (uric acid >  $450 \mu mol/l$ )

## Acute management

- NSAIDs or colchicine are first-line
  - the maximum dose of NSAID should be prescribed until 1-2 days after the symptoms have settled
  - o gastroprotection (e.g. a proton pump inhibitor) may also be indicated
- colchicine
  - inhibits microtubule polymerization by binding to tubulin, interfering with mitosis. Also inhibits neutrophil motility and activity
  - has a slower onset of action
  - may be used with caution in renal impairment: the BNF advises to reduce the dose if eGFR is 10-50 ml/min and to avoid if eGFR < 10 ml/min (BNF)</li>
  - o the main side-effect is diarrhoea
- oral steroids may be considered if NSAIDs and colchicine are contraindicated.
  - o a dose of prednisolone 15mg/day is usually used
- another option is intra-articular steroid injection

• if the patient is already taking allopurinol it should be continued

Indications for urate-lowering therapy (ULT)

- the British Society of Rheumatology Guidelines now advocate offering urate-lowering therapy to all patients after their **first attack of gout**
- ULT is particularly recommended if:
  - > = 2 attacks in 12 months
  - tophi
  - o renal disease
  - uric acid renal stones
  - o prophylaxis if on cytotoxics or diuretics

## Urate-lowering therapy

- it has traditionally been taught that urate-lowering therapy should not be started until 2 weeks after an acute attack, as starting too early may precipitate a further attack. The evidence base to support this however looks weak
- in 2017 the BSR updated their guidelines. They still support a delay in starting urate-lowering therapy because it is better for a patient to make long-term drug decisions whilst not in pain
  - the key passage is: 'Commencement of ULT is best delayed until inflammation has settled as ULT is better discussed when the patient is not in pain'
- allopurinol is first-line
  - $\circ$  initial dose of 100 mg od, with the dose titrated every few weeks to aim for a serum uric acid of < 360  $\mu$ mol/l
  - $\circ$  a lower target uric acid level below 300 µmol/L may be considered for patients who have tophi, chronic gouty arthritis or continue to have ongoing frequent flares despite having a uric acid below 360 µmol/L
  - o a lower initial dose of allopurinol should be given if the patient has a reduced eGFR
  - colchicine cover should be considered when starting allopurinol. NSAIDs can be used if colchicine cannot be tolerated. The BSR guidelines suggest this may need to be continued for 6 months
- the second-line agent when allopurinol is not tolerated or ineffective is febuxostat (also a xanthine oxidase inhibitor)
- in refractory cases other agents may be tried:
  - uricase (urate oxidase) is an enzyme that catalyzes the conversion of urate to the degradation product allantoin. It is present in certain mammals but not humans
  - in patients who have persistent symptomatic and severe gout despite the adequate use
    of urate-lowering therapy, pegloticase (polyethylene glycol modified mammalian uricase)
    can achieve rapid control of hyperuricemia. It is given as an infusion once every two
    weeks

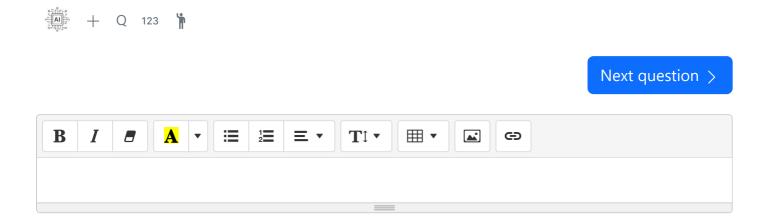
## Lifestyle modifications

- reduce alcohol intake and avoid during an acute attack
- lose weight if obese

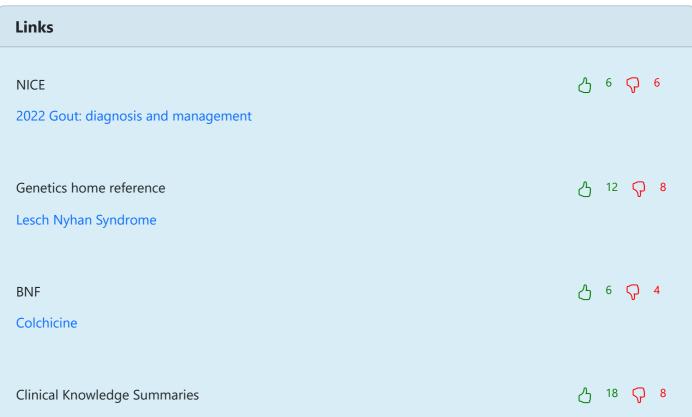
• avoid food high in purines e.g. Liver, kidneys, seafood, oily fish (mackerel, sardines) and yeast products

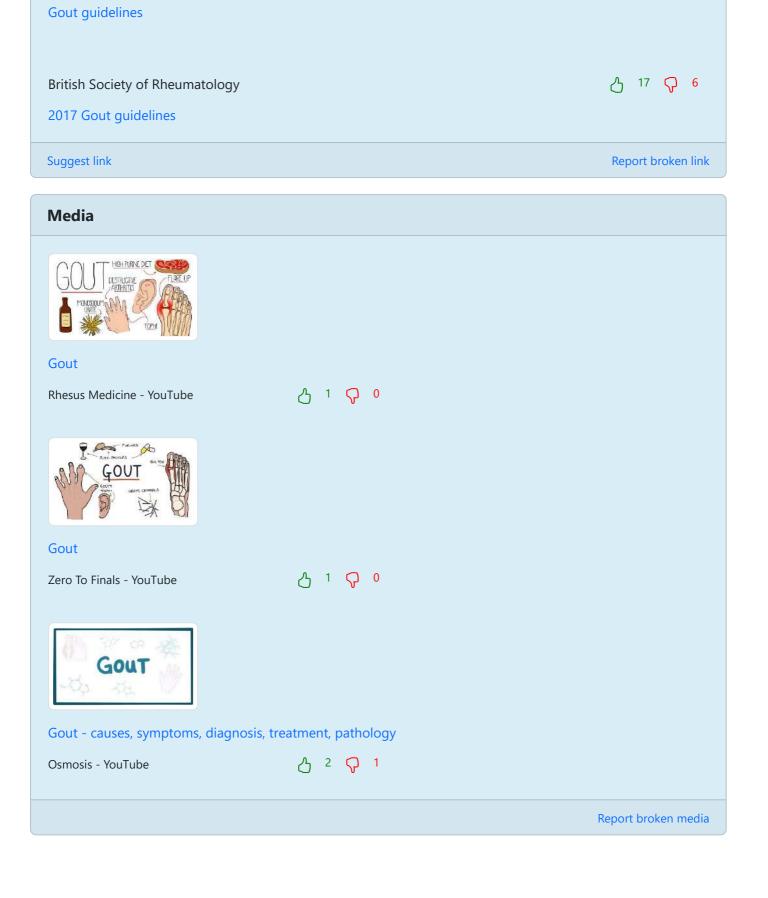
## Other points

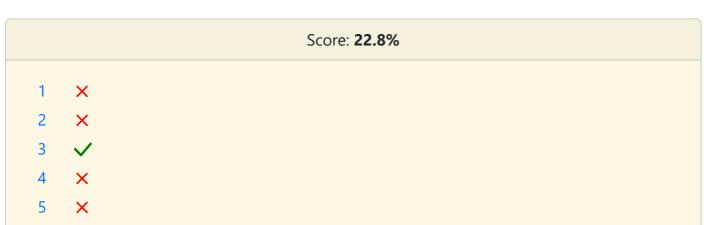
- consideration should be given to stopping precipitating drugs (such as thiazides)
- losartan has a specific uricosuric action and may be particularly suitable for the many patients who have coexistent hypertension
- increased vitamin C intake (either supplements or through normal diet) may also decrease serum uric acid levels











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A 67-year-old female presented to the accident and emergency department with severe headache and shortness of breath for the last six hours followed by seizures which occurred twice during the last hour.

The patient is a known case of diffuse cutaneous systemic sclerosis diagnosed two years ago and she is on steroids and cyclophosphamide.

On examination, she looks ill, agitated and dyspnoeic. Her pulse rate is 100 beats per minute, regular and her blood pressure is 220/110 mmHg.

Her JVP is raised, there is a gallop rhythm and bilateral basal crackles. There is lower limb oedema and brisk reflexes.

Fundoscopy showed grade 3 hypertensive retinopathy.

Investigations done two weeks previously showed:

Serum sodium	140 mmol/L
Serum potassium	5.7 mmol/L
Serum urea	17 mmol/L
Serum creatinine	250 mol/L
Urinalysis	protein ++, blood ++

What is the most appropriate immediate treatment to lower her blood pressure?

IV sodium nitroprusside	
IV labetalol	
Oral ACE inhibitor	
IV hydralazine	
Nitrate infusion	

Submit answer

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Question 24 of 79







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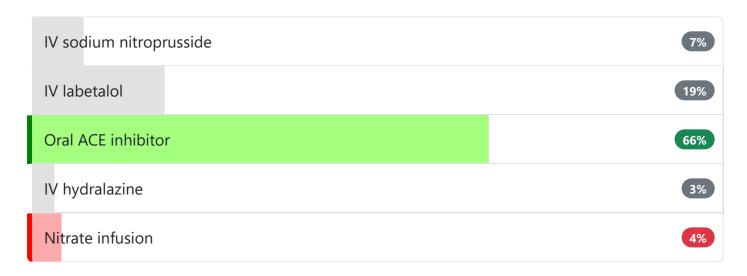
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Serum urea	17 mmol/L
Serum creatinine	250 mol/L
Urinalysis	protein ++, blood ++

What is the most appropriate immediate treatment to lower her blood pressure?



This patient has developed scleroderma renal crisis which presents as malignant hypertension, heart failure and rapid deterioration of renal function progressing to acute renal failure.

This hypertensive emergency should be managed with gradual reduction of blood pressure at a rate of 10-15 mmHg per day with an oral ACE inhibitor as the pathology of scleroderma renal crisis is vasospasm.

IV sodium nitroprusside and IV labetalol should be avoided as they lead to sudden reduction of blood pressure and renal hypoperfusion that leads to acute tubular necrosis, making the already deranged renal function even worse.

Indeed, this patient requires ICU admission for management of her heart failure and acute renal failure.



Next question >

# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - patients with renal disease should be started on an ACE inhibitor
- poor prognosis

Scleroderma (without internal organ involvement)

• tightening and fibrosis of skin

• may be manifest as plaques (morphoea) or linear



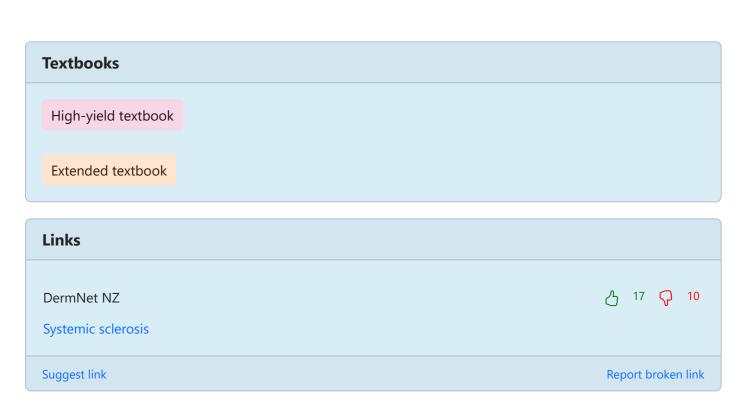


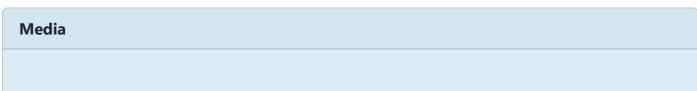


## **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis









### Scleroderma

Townsend Teaching - YouTube 4 Q 0









## Scleroderma

Osmosis - YouTube











# Systemic Sclerosis and Scleroderma

Zero to Finals - YouTube







Report broken media

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Question 25 of 79





A 35-year-old woman presents to rheumatology clinic with a 2-month history of symmetrical swelling of the ankles and fingers. She also complains of joint pain and stiffness. The stiffness is primarily worse in the early morning and eases with use. Apart from a recent sore throat, she is otherwise well. She has a family history of type 1 diabetes mellitus. She does not take any prescribed medication but has found herself relying on over-the-counter analgesics to get through the day.

On examination, she has bilateral swelling of the index, ring and middle fingers and bilateral ankle swelling. She has a full range of movement in the fingers, wrists and ankles. There is marked swelling and tenderness to palpation at the distal interphalangeal joints in the index, middle and ring fingers on both sides. There are no skin changes, but yellowing and pitting of the nails are noted.

#### Blood tests show:

Hb	11.1 g/dl
Platelets	305 * 10 <sup>9</sup> /l
WBC	7.8 * 10 <sup>9</sup> /l

Na <sup>+</sup>	141 mmol/l
K <sup>+</sup>	4.2 mmol/l
Urea	5.8 mmol/l
Creatinine	64 µmol/l

Bilirubin	13 µmol/l
ALP	83 u/l
ALT	15 u/l
ESR	50mm/hr
CRP	39 mg/L
Rheumatoid factor	negative

Hand X-ray shows mild erosion at the distal interphalangeal joints of the index, middle and ring fingers on both hands.

What is the diagnosis?

Rheumatoid arthritis	
Reiters syndrome	×
Ankylosing spondylitis	×
Yellow nail syndrome	×
Psoriatic arthritis	

# Submit answer

Reference ranges  $\checkmark$ 

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Question 25 of 79







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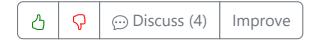
Hand X-ray shows mild erosion at the distal interphalangeal joints of the index, middle and ring fingers on both hands.

What is the diagnosis?

Rheumatoid arthritis	7%
Reiters syndrome	3%
Ankylosing spondylitis	1%
Yellow nail syndrome	9%
Psoriatic arthritis	80%

Although the patient does not have a psoriatic rash, she has classic symptoms of psoriatic arthritis. She has dactylitis and distal interphalangeal swelling, as well as ankle involvement. Nail signs are well-documented in psoriasis. The diagnosis is clinched by the negative rheumatoid factor and raised inflammatory markers. A slightly low haemoglobin is also a common feature of the disease. The pattern of joint involvement points more towards psoriatic arthritis than rheumatoid arthritis, in which the metacarpophalangeal joints and wrists are more commonly affected. Reiter's syndrome is a reactive arthritis that typically follows a gastrointestinal or venereal infection. Conjunctivitis and urethritis are seen alongside arthritis. Back pain would be expected to accompany ankylosing spondylitis. Yellow nail syndrome is a rare disorder of uncertain pathogenesis. It presents with nail discolouration, lymphoedema and pleural effusions.

Psoriatic arthritis can manifest in the absence of skin signs, particularly if the patient has a family history of psoriasis. The patient may develop a rash later or have signs limited to the nails.



Next question >

# Psoriatic arthropathy \*

Psoriatic arthropathy is an inflammatory arthritis associated with psoriasis and is classed as one of the seronegative spondyloarthropathies. It correlates poorly with cutaneous psoriasis and often precedes the development of skin lesions. Around 10-20% of patients with skin lesions develop an arthropathy with males and females being equally affected.

# **Presentation**

#### **Patterns**

- symmetric polyarthritis
  - very similar to rheumatoid arthritis
  - 30-40% of cases, most common type
- asymmetrical oligoarthritis: typically affects hands and feet (20-30%)

- until recently it was thought asymmetrical oligoarthritis was the most common type, based on data from the original 1973 Moll and Wright paper. Please see the link for a comparison of more recent studies
- sacroiliitis
- DIP joint disease (10%)
- arthritis mutilans (severe deformity fingers/hand, 'telescoping fingers')

# Other signs

- psoriatic skin lesions
- periarticular disease tenosynovitis and soft tissue inflammation resulting in:
  - enthesitis: inflammation at the site of tendon and ligament insertion e.g. Achilles tendonitis, plantar fascitis
  - o tenosynovitis: typically of the flexor tendons of the hands
  - o dactylitis: diffuse swelling of a finger or toe
- nail changes
  - pitting
  - onycholysis

# **Investigation and management**

## X-ray

- often have the unusual combination of coexistence of erosive changes and new bone formation
- periostitis
- 'pencil-in-cup' appearance

### Management

- should be managed by a rheumatologist
- treatment is similar to that of rheumatoid arthritis (RA). However, the following differences are noted:
  - mild peripheral arthritis/mild axial disease may be treated with 'just' an NSAID, rather than all patients being on disease-modifying therapy as with RA
  - o if more moderate/severe disease then methotrexate is typically used as in RA
  - use of monoclonal antibodies such as ustekinumab (targets both IL-12 and IL-23) and secukinumab (targets IL-17)
  - o apremilast: phosphodiesterase type-4 (PDE4) inhibitor → suppression of proinflammatory mediator synthesis and promotion of anti-inflammatory mediators
  - o has a better prognosis than RA











Next question >



# **Textbooks**

High-yield textbook

Extended textbook

## Links

Annals of the Rheumatic Diseases

占 10 ♀ 11

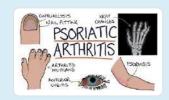
Relative incidence of polyarthritis vs. oligoarthritis

Patient.info

Psoriatic arthropathy review

Suggest link Report broken link

# Media



Psoriatic arthritis

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## Question 26 of 79





A 28-year-old woman with systemic lupus erythematosus attends the pre-conception clinic. She would like some advice regarding her medications prior to getting pregnant. She has never been pregnant before and her lupus has been stable on her current medications: mycophenolate and hydroxychloroquine for over 12 months. She also has asthma, which is well controlled with beclomethasone and salbutamol inhalers, and she takes regular omeprazole for gastro-oesophageal reflux.

What is the most appropriate medication amendment?

Half omeprazole dose	
Stop beclomethasone inhaler	
Stop hydroxycholorquine	
Add ramipril 1.25mg	
Change mycophenolate to azathioprine	

Submit answer

Reference ranges ∨

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Question 26 of 79



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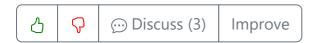
A 28-year-old woman with systemic lupus erythematosus attends the pre-conception clinic. She would like some advice regarding her medications prior to getting pregnant. She has never been pregnant before and her lupus has been stable on her current medications: mycophenolate and hydroxychloroquine for over 12 months. She also has asthma, which is well controlled with beclomethasone and salbutamol inhalers, and she takes regular omeprazole for gastro-oesophageal reflux.

What is the most appropriate medication amendment?



Unlike the majority of autoimmune conditions, pregnancy increases the likelihood of a lupus flare. It is essential that a patients lupus is well controlled and quiescent for at least six months prior to pregnancy. Given that mycophenolate is teratogenic in pregnancy, it must be stopped. It is common practice to change to azathioprine, which has been shown to be safe in pregnancy - but still used with caution! However knowledge of common medications used/not used in pregnancy should also lead the candidate to this option.

There is no need to stop the beclomethasone or hydroxycholoquine, as they are both safe in pregnancy. Omeprazole is also safe in pregnancy and the dose does not need to be changed. ACE inhibitors are contraindicated in pregnancy and must be stopped/changed to another agent.



Next question >

# Systemic lupus erythematosus: management \*

# Basics

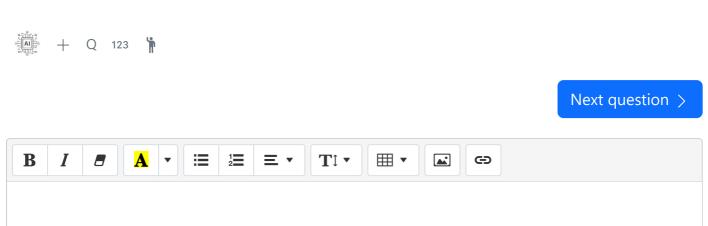
NSAIDs

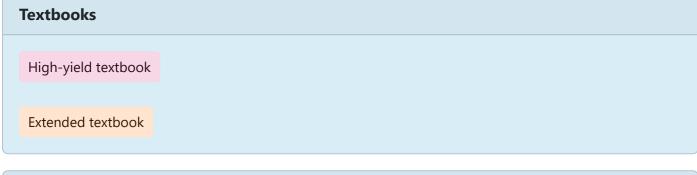
• sun-block

## Hydroxychloroquine

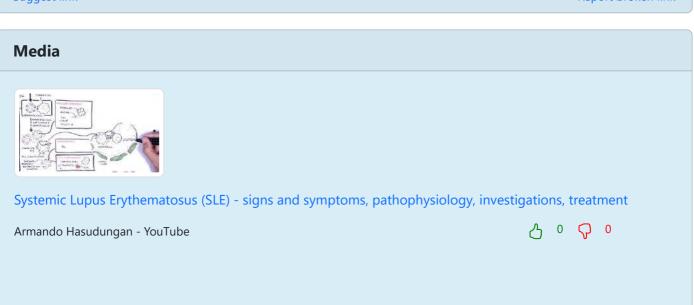
• the treatment of choice for SLE

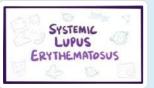
If internal organ involvement e.g. renal, neuro, eye then consider prednisolone, cyclophosphamide











Systemic lupus erythematosus (SLE) - causes, symptoms, diagnosis & pathology

Osmosis - YouTube







Report broken media

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#### Question 27 of 79





A 58-year-old gentleman of Afro-Caribbean origin presents to clinic. He complains of lethargy, fever and joint pain, particularly in both knees. He has also noticed an intermittent rash on his arms and his neck. These symptoms have progressed over the last six months. He has a past medical history of congestive cardiac failure and chronic renal failure secondary to hypertension. His medication includes bisoprolol, aspirin and hydralazine/isosorbide dinitrate.

On examination the patient has no evidence of joint swelling. He has discrete patches of scaling on his neck and forearms. He has normal power in his arms and legs.

The following blood tests were obtained;

Rheumatoid Factor	Positive
Anti-nuclear antibody	Positive
Anti-single stranded DNA	Positive
Anti-extractable nuclear Antigen	Negative
Anti-histone antibodies	Positve

What is the most likely diagnosis?

Polymorphous Light Eruption	
Dermatomyositis	
Drug induced lupus erythematous	
Rheumatoid arthritis	
Systemic lupus erythematosus	

Submit answer

Reference ranges ∨

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Question 27 of 79



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The following blood tests were obtained;

Rheumatoid Factor	Positive
Anti-nuclear antibody	Positive
Anti-single stranded DNA	Positive
Anti-extractable nuclear Antigen	Negative
Anti-histone antibodies	Positve

What is the most likely diagnosis?



A polymorphous rash would expect to be erythematous and less associated with systemic symptoms. Dermatomyositis may have a positive ENA (anti-Jo1) with heliotrope rash and Gottrons papulses. There also may be associated muscle weakness. Rheumatoid arthritis would expect to show arthritis and is less associated with a rash and the antibody picture. SLE is more associated with double stranded DNA and ENA.

Drug induced lupus can be caused by a variety of drugs but most commonly antihypertensives and antifungals. Specific examples include isoniazid, hydralazine, procainamide, diltiazem and

phenytoin. Anti-histone antibodies are associated with drug-induced lupus in most cases.



Next question >

# Drug-induced lupus \*

In drug-induced lupus not all the typical features of systemic lupus erythematosus are seen, with renal and nervous system involvement being unusual. It usually resolves on stopping the drug.

#### **Features**

- arthralgia
- myalgia
- skin (e.g. malar rash) and pulmonary involvement (e.g. pleurisy) are common
- ANA positive in 100%, dsDNA negative
- anti-histone antibodies are found in 80-90%
- anti-Ro, anti-Smith positive in around 5%



© Image used on license from DermNet NZ

A woman with drug-induced lupus

#### Most common causes

- procainamide
- hydralazine

#### Less common causes

isoniazid

- minocycline
- phenytoin

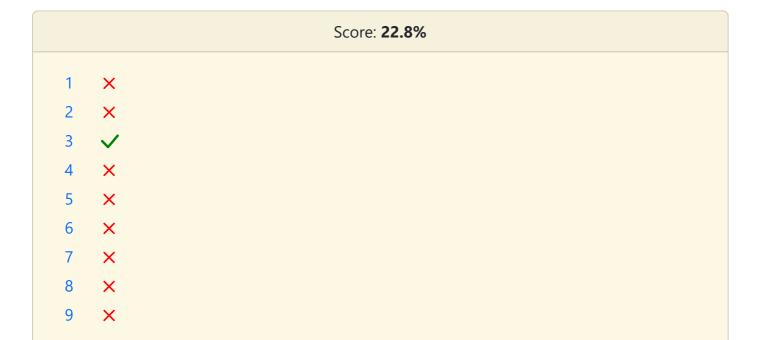


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Question 28 of 79

P



An 18 year-old girl with neuropsychiatric Wilsons disease is commenced on chelation therapy.

One month later she presents with fatigue, generalised arthralgia, and an erythematous rash affecting the face, back of the neck, shoulders, and arms.

Which test is most specific for the suspected diagnosis?

Anti-centromere antibodies	
Anti-Scl70 antibodies	
Anti-mitochondrial antibodies	
Anti-histone antibodies	
Anti-dsDNA antibodies	

## Submit answer

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Question 28 of 79

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 $\Rightarrow$ 

An 18 year-old girl with neuropsychiatric Wilsons disease is commenced on chelation therapy.

One month later she presents with fatigue, generalised arthralgia, and an erythematous rash affecting the face, back of the neck, shoulders, and arms.

Which test is most specific for the suspected diagnosis?



This is drug-induced lupus as a result of penicillamine chelation therapy.

Anti-histone antibodies are specific for drug-induced lupus.

Other unusual side-effects of penicillamine include membranous glomerulonephritis and a myasthenia-like syndrome.

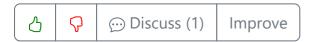
Other causes of drug-induced lupus include procainamide, minocycline, hydralazine, and isoniazid.

Anti-centromere antibodies are found in limited systemic sclerosis.

Anti-Scl70 antibodies are found in diffuse systemic sclerosis.

Anti-mitochondrial antibodies are specific for primary biliary cirrhosis.

Anti-dsDNA antibodies are specific for systemic lupus erythematosus.



# Drug-induced lupus \*

In drug-induced lupus not all the typical features of systemic lupus erythematosus are seen, with renal and nervous system involvement being unusual. It usually resolves on stopping the drug.

#### **Features**

- arthralgia
- myalgia
- skin (e.g. malar rash) and pulmonary involvement (e.g. pleurisy) are common
- ANA positive in 100%, dsDNA negative
- anti-histone antibodies are found in 80-90%
- anti-Ro, anti-Smith positive in around 5%



A woman with drug-induced lupus

#### Most common causes

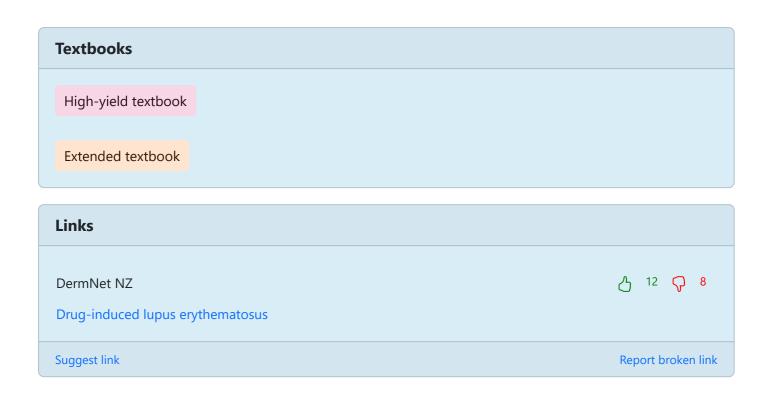
- procainamide
- hydralazine

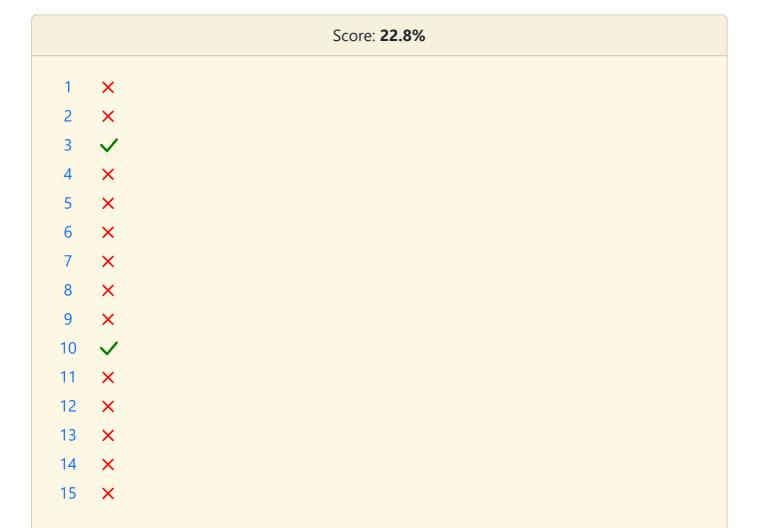
#### Less common causes

- isoniazid
- minocycline
- phenytoin









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Question 29 of 79





A 52-year-old man presents with lethargy and reduced sensation in both feet. He reports a 2 month history of fevers and 4kg weight loss. He also reports intermittent testicular pain.

On examination there is livedo reticularis on both legs and reduced light touch and pain sensation on both feet.

#### Blood tests reveal:

Hb	116 g/l	Na <sup>+</sup>	137 mmol/l	Bilirubin	18 µmol/l
Platelets	487 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.8 mmol/l	ALP	92 u/l
WBC	8.3 * 10 <sup>9</sup> /l	Urea	12.8 mmol/l	ALT	102 u/l
Neuts	6.3 * 10 <sup>9</sup> /l	Creatinine	182 µmol/l	γGT	16 u/l
MCV	89 fL	ESR	78mm/hr	Albumin	35 g/l

What is the most likely diagnosis?

Cryoglobulinaemia	
Granulomatosis with polyangiitis	
Eosinophilic granulomatosis with polyangiitis	
Polyarteritis nodosa	
Microscopic polyangiitis	

Submit answer

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Question 29 of 79



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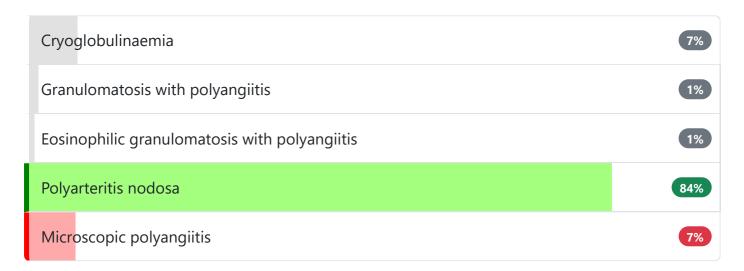
A 52-year-old man presents with lethargy and reduced sensation in both feet. He reports a 2 month history of fevers and 4kg weight loss. He also reports intermittent testicular pain.

On examination there is livedo reticularis on both legs and reduced light touch and pain sensation on both feet.

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Hb	116 g/l	Na <sup>+</sup>	137 mmol/l	Bilirubin	18 µmol/l
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WBC	8.3 * 10 <sup>9</sup> /l	Urea	12.8 mmol/l	ALT	102 u/l
Neuts	6.3 * 10 <sup>9</sup> /l	Creatinine	182 µmol/l	γGT	16 u/l
MCV	89 fL	ESR	78mm/hr	Albumin	35 g/l

What is the most likely diagnosis?



Fever, lethargy, neuropathy, testicular pain and renal dysfunction are consistent with polyarteritis nodosa (PAN).

All systemic vasculitides can present with fever and lethargy, however polyarteritis nodosa is the systemic vasculitis most frequently associated with testicular involvement.

Hernández-RodrÃguez, J., Tan, C.D., Koening, C.L., Khasnis, A., RodrÃguez, E.R. and Hoffman, G.S., 2012. Testicular vasculitis: findings differentiating isolated disease from systemic disease in 72 patients. Medicine, 91(2), pp.75-85.

# Polyarteritis nodosa 🖈

Polyarteritis nodosa (PAN) is a vasculitis affecting medium-sized arteries with necrotizing inflammation leading to aneurysm formation. PAN is more common in middle-aged men and is associated with hepatitis B infection.

#### **Features**

- fever, malaise, arthralgia
- weight loss
- hypertension
- mononeuritis multiplex, sensorimotor polyneuropathy

Improve

- testicular pain
- livedo reticularis
- haematuria, renal failure
- perinuclear-antineutrophil cytoplasmic antibodies (ANCA) are found in around 20% of patients with 'classic' PAN
- hepatitis B serology positive in 30% of patients



© Image used on license from DermNet NZ



© Image used on license from Radiopaedia

Angiogram from a patient with polyarteritis nodosa. Both kidneys demonstrate beading and numerous microaneurysms affecting the intrarenal vessels. Similar changes are seen affecting the intrahepatic vessels with a few small microaneurysms noted. The proximal branches of the SMA appears normal; however there are no normal straight arteries from the jejunal arteries and lack of normal anastomotic arcades and loops. This is associated with multiple microaneurysms.

Next question >



## **Textbooks**

High-yield textbook

Extended textbook

#### Media



Polyarteritis Nodosa and Kawasaki Disease (Medium Vessel Vasculitis) - Symptoms, pathophysiology

Armando Hasudungan - YouTube

Report broken media

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Question 30 of 79





A 40-year-old man is investigated for back pain. For the past few months he has been troubled with pain in his lower back which is typically worse in the morning and better by the end of the day. There is some radiation of pain to the right buttock but no leg pains. An x-ray of his lumbar spine is shown below



© Image used on license from Radiopaedi

What is the most likely cause of his back pain?

Lumbar disc prolapse at multiple levels	
Osteopetrosis	
Calcification of the vertebral artery	
Spinal stenosis	
Ankylosing spondylitis	

# Submit answer

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Question 30 of 79







A 40-year-old man is investigated for back pain. For the past few months he has been troubled with pain in his lower back which is typically worse in the morning and better by the end of the day. There is some radiation of pain to the right buttock but no leg pains. An x-ray of his lumbar spine is shown below



© Image used on license from Radionaedia



## What is the most likely cause of his back pain?



This image shows the typical appearance of bamboo spine with a single central radiodense line related to ossification of supraspinous and interspinous ligaments which is called dagger sign. Ankylosing is detectable in both sacroiliac joints.

Note the history of morning pain is typical for an inflammatory arthritis such as ankylosing spondylitis.



Next question >

# Ankylosing spondylitis: investigation and management \*



Ankylosing spondylitis is a HLA-B27 associated spondyloarthropathy. It typically presents in males (sex ratio 3:1) aged 20-30 years old.

# Investigation

Inflammatory markers (ESR, CRP) are typically raised although normal levels do not exclude ankylosing spondylitis.

HLA-B27 is of little use in making the diagnosis as it is positive in:

- 90% of patients with ankylosing spondylitis
- 10% of normal patients

Plain x-ray of the sacroiliac joints is the most useful investigation in establishing the diagnosis. Radiographs may be normal early in disease, later changes include:

- sacroiliitis: subchondral erosions, sclerosis
- squaring of lumbar vertebrae
- 'bamboo spine' (late & uncommon)
- syndesmophytes: due to ossification of outer fibers of annulus fibrosus
- chest x-ray: apical fibrosis

If the x-ray is negative for sacroiliac joint involvement in ankylosing spondylitis but suspicion for AS remains high, the next step in the evaluation should be obtaining an MRI. Signs of early inflammation involving sacroiliac joints (bone marrow oedema) confirm the diagnosis of AS and prompt further treatment.

Spirometry may show a restrictive defect due to a combination of pulmonary fibrosis, kyphosis and ankylosis of the costovertebral joints.













# Management

The following is partly based on the 2010 EULAR guidelines (please see the link for more details):

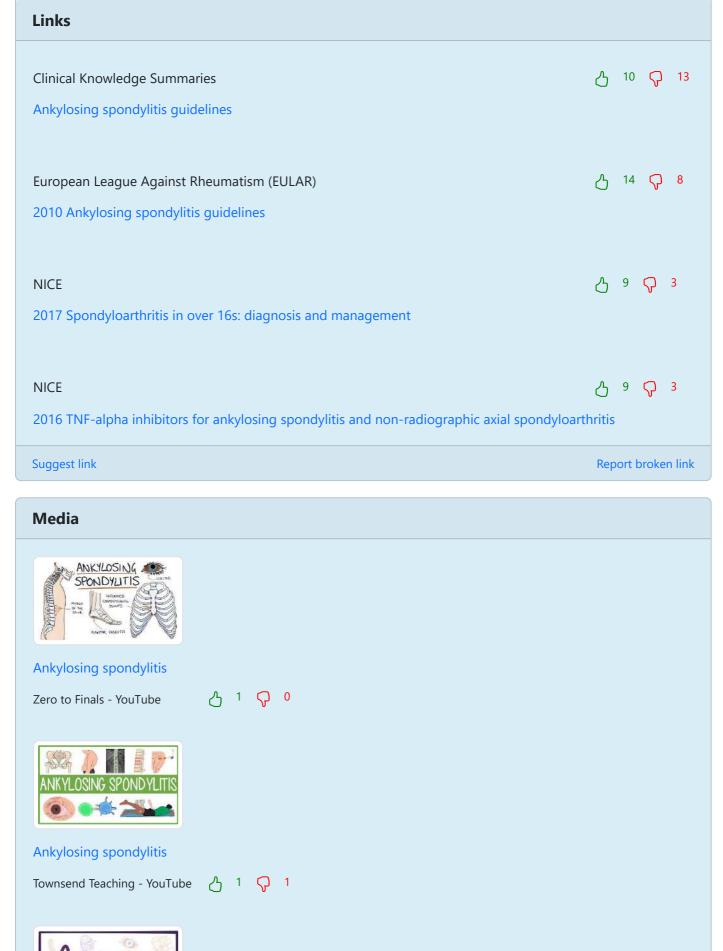
- encourage regular exercise such as swimming
- NSAIDs are the first-line treatment
- physiotherapy
- the disease-modifying drugs which are used to treat rheumatoid arthritis (such as sulphasalazine) are only really useful if there is peripheral joint involvement
- the 2010 EULAR guidelines suggest: 'Anti-TNF therapy should be given to patients with persistently high disease activity despite conventional treatments'
- research is ongoing to see whether anti-TNF therapies such as etanercept and adalimumab should be used earlier in the course of the disease



Next question >









#### Ankylosing spondylitis

Osmosis - YouTube







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A 26-year-old woman presents with a four month history of back pain. The pain is located around the lower lumbar vertebrae and spreads to both buttocks. Ibuprofen and walking seem to improve the pain. A lumbar spine film is requested:



What is the most likely cause of this patients back pain?

Marble bone disease	
Discitis	
Ankylosing spondylitis	
Facet-joint dysfunction	
Rheumatoid arthritis	

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Question 31 of 79

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A 26-year-old woman presents with a four month history of back pain. The pain is located around the lower lumbar vertebrae and spreads to both buttocks. Ibuprofen and walking seem to improve the pain. A lumbar spine film is requested:



What is the most likely cause of this patients back pain?



Ankylosing spondylitis with well formed syndesmophytes are seen on the lumbar spine film.

The first thing to address is the sex of the patient. Of course ankylosing spondylitis is more common in men but the male-to-female ratio is only 3:1. This means it is reasonable to be asked about female patients in questions, particularly if there is accompanying 'hard evidence' such as x-

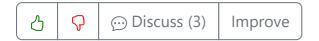
rays.

Marble bone disease (osteopetrosis) results in dense, thick bones that are prone to fracture. Syndesmophytes are not a feature.

Facet-joint dysfunction is a common cause of back pain but it would not explain the x-ray findings.

Rheumatoid arthritis of course does not commonly present with back pain. The following x-ray changes are typically seen:

- loss of joint space
- juxta-articular osteoporosis
- soft-tissue swelling
- periarticular erosions
- subluxation



Next question >

# Ankylosing spondylitis: investigation and management



Ankylosing spondylitis is a HLA-B27 associated spondyloarthropathy. It typically presents in males (sex ratio 3:1) aged 20-30 years old.

# Investigation

Inflammatory markers (ESR, CRP) are typically raised although normal levels do not exclude ankylosing spondylitis.

HLA-B27 is of little use in making the diagnosis as it is positive in:

- 90% of patients with ankylosing spondylitis
- 10% of normal patients

Plain x-ray of the sacroiliac joints is the most useful investigation in establishing the diagnosis. Radiographs may be normal early in disease, later changes include:

- sacroiliitis: subchondral erosions, sclerosis
- squaring of lumbar vertebrae
- 'bamboo spine' (late & uncommon)
- syndesmophytes: due to ossification of outer fibers of annulus fibrosus
- chest x-ray: apical fibrosis

If the x-ray is negative for sacroiliac joint involvement in ankylosing spondylitis but suspicion for AS remains high, the next step in the evaluation should be obtaining an MRI. Signs of early inflammation involving sacroiliac joints (bone marrow oedema) confirm the diagnosis of AS and prompt further treatment.

Spirometry may show a restrictive defect due to a combination of pulmonary fibrosis, kyphosis and ankylosis of the costovertebral joints.













# Management

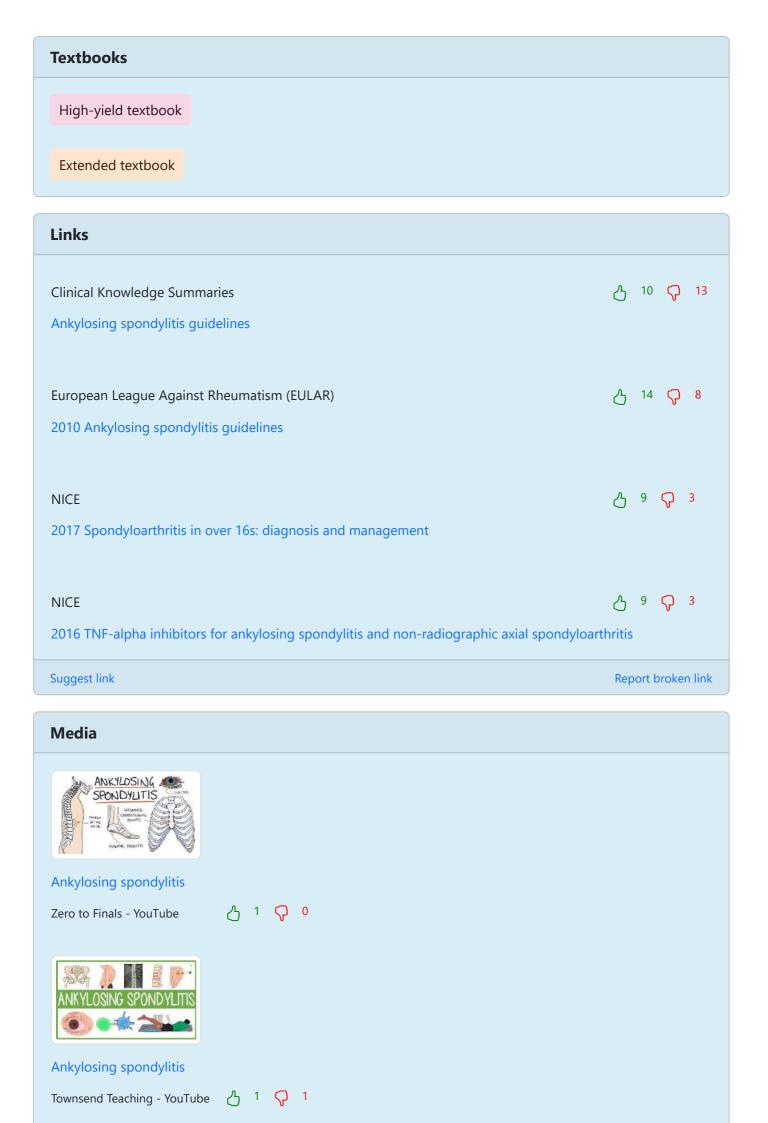
The following is partly based on the 2010 EULAR guidelines (please see the link for more details):

- encourage regular exercise such as swimming
- NSAIDs are the first-line treatment
- physiotherapy
- the disease-modifying drugs which are used to treat rheumatoid arthritis (such as sulphasalazine) are only really useful if there is peripheral joint involvement
- the 2010 EULAR guidelines suggest: 'Anti-TNF therapy should be given to patients with persistently high disease activity despite conventional treatments'
- research is ongoing to see whether anti-TNF therapies such as etanercept and adalimumab should be used earlier in the course of the disease



Next question >







## Ankylosing spondylitis

Osmosis - YouTube





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## Question 32 of 79





A 56-year-old man presents with buttock pain. This has been present for many years but has recently become worse. The pain is usually worse in the early part of the day and often eases by the late afternoon. An x-ray is requested:



## What is the most likely underlying diagnosis?

Ankylosing spondylitis	
Alkaptonuria	
Multiple myeloma	
Peripheral arterial disease	
Bilateral hip osteoarthritis	

Submit answer

Reference ranges ✓

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Question 32 of 79



 $\Box$ 



A 56-year-old man presents with buttock pain. This has been present for many years but has recently become worse. The pain is usually worse in the early part of the day and often eases by the late afternoon. An x-ray is requested:



© Image used on license from Radiopaedia

What is the most likely underlying diagnosis?



Fusion of bilateral sacroiliac joints consistent with ankylosing spondylitis. Sacroiliitis may present as sclerosis of joint margins which can be asymmetrical at early stage of disease, but is bilateral and symmetrical in late disease.

Pain that is worse in the morning is consistent with an inflammatory condition such as ankylosing spondylitis.





Ankylosing spondylitis is a HLA-B27 associated spondyloarthropathy. It typically presents in males (sex ratio 3:1) aged 20-30 years old.

# Investigation

Inflammatory markers (ESR, CRP) are typically raised although normal levels do not exclude ankylosing spondylitis.

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Spirometry may show a restrictive defect due to a combination of pulmonary fibrosis, kyphosis and ankylosis of the costovertebral joints.









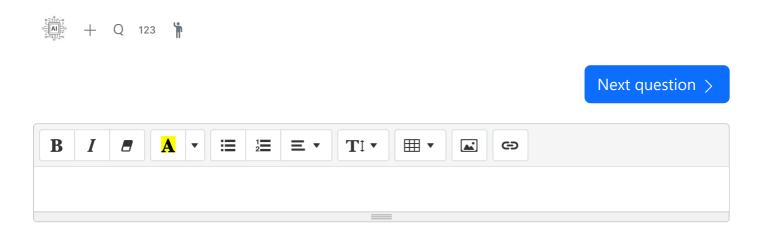




## Management

The following is partly based on the 2010 EULAR guidelines (please see the link for more details):

- encourage regular exercise such as swimming
- NSAIDs are the first-line treatment.
- physiotherapy
- the disease-modifying drugs which are used to treat rheumatoid arthritis (such as sulphasalazine) are only really useful if there is peripheral joint involvement
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- research is ongoing to see whether anti-TNF therapies such as etanercept and adalimumab should be used earlier in the course of the disease





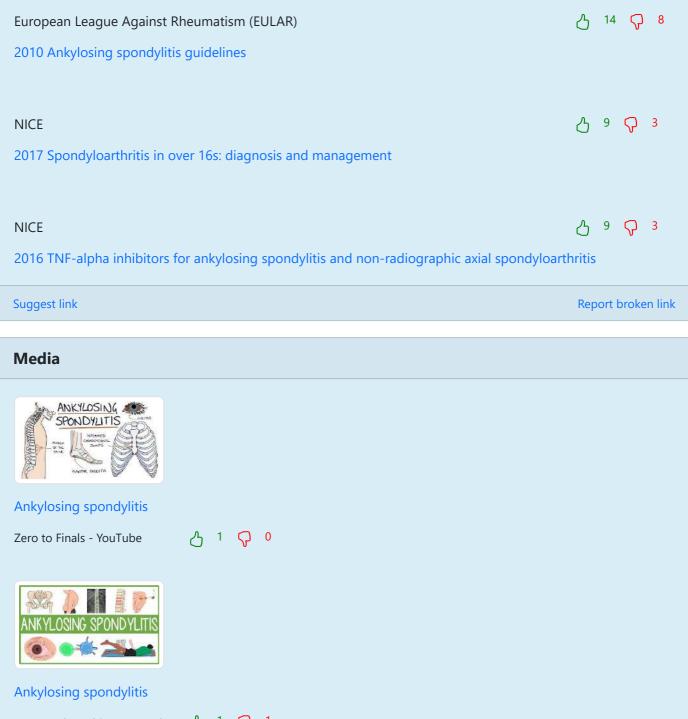


Clinical Knowledge Summaries









Townsend Teaching - YouTube <a>↑ 1</a> <a>↑ 1</a> <a>↑ 1</a>



#### Ankylosing spondylitis

Osmosis - YouTube  $\bigcirc$  0  $\bigcirc$  1

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Question 33 of 79





A 36-year-old man presents with progressive lower back pain for the past six months. The pain is worse in the mornings and tends to ease with exercise and the passage of the day. He has tried paracetamol but this does not fully controlled his pain. An x-ray of his spine is shown below:



## What is the most appropriate first-line treatment

Methotrexate	
Sulfasalazine	
Naproxen	
Vitamin D supplementation	
Infliximab	

# Submit answer

Reference ranges  $\vee$ 

# Score: 0%

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Back to top





Question 33 of 79



 $\Box$ 



A 36-year-old man presents with progressive lower back pain for the past six months. The pain is worse in the mornings and tends to ease with exercise and the passage of the day. He has tried paracetamol but this does not fully controlled his pain. An x-ray of his spine is shown below:



## What is the most appropriate first-line treatment



The x-ray shows syndesmophytes and squaring of vertebral bodies consistent with a diagnosis of

ankylosing spondylitis.

NSAIDs are the first-line drug treatment in an ankylosing spondylitis. Regular exercise is also very important. The role of anti-TNF therapies is increasing but they are not currently first-line and the 2008 NICE guidelines specifically advise against using infliximab.



Next question >

# Ankylosing spondylitis: investigation and management \*

Ankylosing spondylitis is a HLA-B27 associated spondyloarthropathy. It typically presents in males (sex ratio 3:1) aged 20-30 years old.

# Investigation

Inflammatory markers (ESR, CRP) are typically raised although normal levels do not exclude ankylosing spondylitis.

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- 'bamboo spine' (late & uncommon)
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# Management

The following is partly based on the 2010 EULAR guidelines (please see the link for more details):

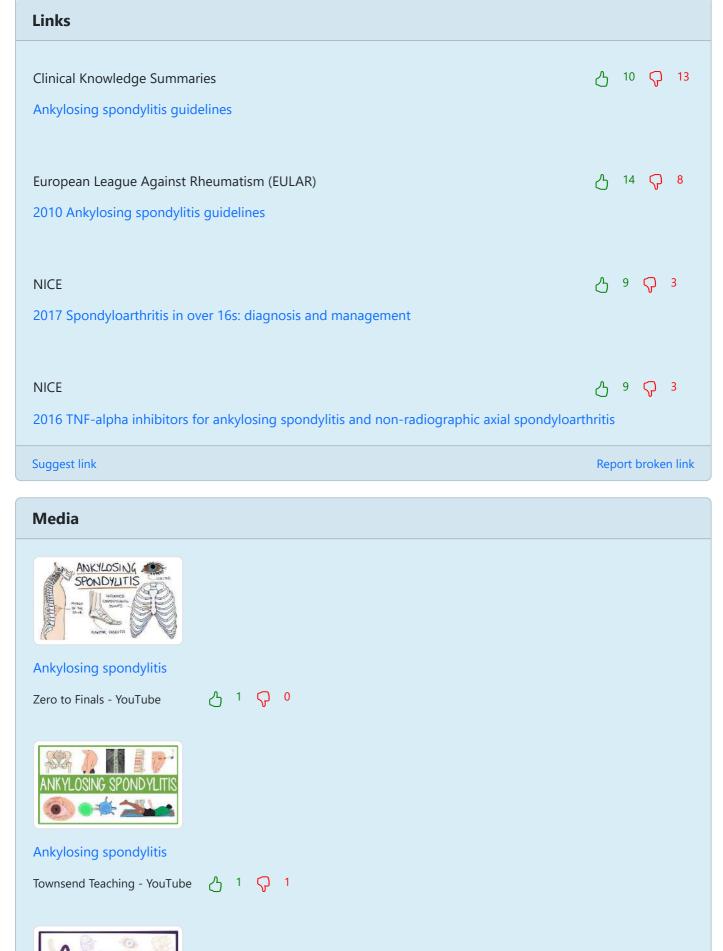
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- NSAIDs are the first-line treatment
- physiotherapy
- the disease-modifying drugs which are used to treat rheumatoid arthritis (such as sulphasalazine) are only really useful if there is peripheral joint involvement
- the 2010 EULAR guidelines suggest: 'Anti-TNF therapy should be given to patients with persistently high disease activity despite conventional treatments'
- research is ongoing to see whether anti-TNF therapies such as etanercept and adalimumab should be used earlier in the course of the disease



Next question >









#### Ankylosing spondylitis

Osmosis - YouTube







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Question 34 of 79





A 78-year-old man is investigated for headaches. A routine blood screen is normal other than an elevated ALP. A skull x-ray is ordered:



## What is the most likely diagnosis?

Myeloma	
Cervical spondylosis	
Pituitary tumour	
Calcified temporal arteritis	
Paget's disease of the bone	

Submit answer

Reference ranges  $\vee$ 







Question 34 of 79



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A 78-year-old man is investigated for headaches. A routine blood screen is normal other than an elevated ALP. A skull x-ray is ordered:

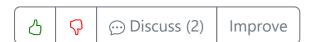


#### What is the most likely diagnosis?



This is a very stereotypical question - you should always think Paget's disease if shown a skull x-ray in an exam.

The radiograph demonstrates marked thickening of the calvarium. There are also ill-defined sclerotic and lucent areas throughout. These features are consistent with Paget's disease.



# Paget's disease of the bone \*

Paget's disease is a disease of increased but uncontrolled bone turnover. It is thought to be primarily a disorder of osteoclasts, with excessive osteoclastic resorption followed by increased osteoblastic activity. Paget's disease is common (UK prevalence 5%) but symptomatic in only 1 in 20 patients. The skull, spine/pelvis, and long bones of the lower extremities are most commonly affected.

### Predisposing factors

- increasing age
- male sex
- northern latitude
- family history

### Clinical features - only 5% of patients are symptomatic

- the stereotypical presentation is an older male with bone pain and an isolated raised ALP
- bone pain (e.g. pelvis, lumbar spine, femur)
- classical, untreated features: bowing of tibia, bossing of skull

### Investigations

- bloods
  - raised alkaline phosphatase (ALP)
  - o calcium and phosphate are typically normal. Hypercalcaemia may occasionally occur with prolonged immobilisation
- other markers of bone turnover include
  - procollagen type I N-terminal propeptide (PINP)
  - serum C-telopeptide (CTx)
  - urinary N-telopeptide (NTx)
  - urinary hydroxyproline
- x-rays
  - osteolysis in early disease → mixed lytic/sclerotic lesions later
  - o skull x-ray: thickened vault, osteoporosis circumscripta
- bone scintigraphy
  - o increased uptake is seen focally at the sites of active bone lesions

#### Management

- indications for treatment include
  - bone pain
  - skull or long bone deformity
  - fracture



- o periarticular Paget's
- bisphosphonate (either oral risedronate or IV zoledronate)
- calcitonin is less commonly used now

## Complications

- deafness (cranial nerve entrapment)
- bone sarcoma (1% if affected for > 10 years)
- fractures
- skull thickening
- high-output cardiac failure



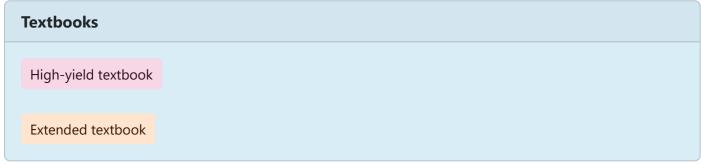


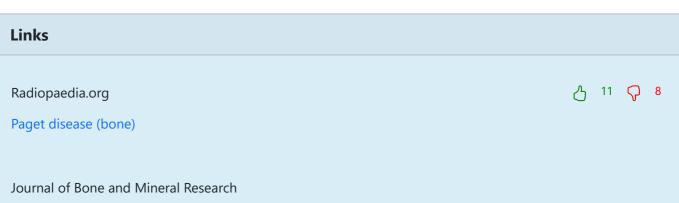




Next question >







## 2019 Paget's disease of the bone guidelines

Suggest link Report broken link

## Media



## Paget's Disease of the bone

Armando Hasudungan - YouTube  $\bigcirc$  0  $\bigcirc$  0









## Paget's disease of the bone

Osmosis - YouTube





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Question 35 of 79

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An 85-year-old woman presents with a long history of poorly controlled type 2 diabetes mellitus presents to her GP complaining of a swollen right foot. She describes it as a 'gammy' foot and says she is always tripping over it. The pain is described as being 2 out of 10. The patient also describes reduced sensation up to her ankles.

On examination she has reduced sensation in both feet. The right midfoot is swollen, warm and slightly erythematous but there is no ulcer present. The dorsalis pedis pulse is difficult to feel on the right hand side.

An x-ray is requested:



# What is the most likely diagnosis?

Septic arthritis of the 1st metatarsophalangeal joint	
Osteomyelitis	
Charcot joint	
Critical ischaemia of the right foot secondary to peripheral arterial disease	

	Gout						
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Question 35 of 79



 $\Box$ 



An 85-year-old woman presents with a long history of poorly controlled type 2 diabetes mellitus presents to her GP complaining of a swollen right foot. She describes it as a 'gammy' foot and says she is always tripping over it. The pain is described as being 2 out of 10. The patient also describes reduced sensation up to her ankles.

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An x-ray is requested:



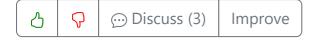
© Image used on license from Radiopaedia

# What is the most likely diagnosis?

Septic arthritis of the 1st metatarsophalangeal joint	1%
Osteomyelitis	10%
Charcot joint	81%
Critical ischaemia of the right foot secondary to peripheral arterial disease	5%

The x-ray shows extensive bone remodeling / fragmentation involving the midfoot. In combination with the presence of a swollen, red, warm joint in a patient with a history of poorly controlled diabetes is highly suggestive of a Charcot's joint.

The x-ray findings are not consistent with osteomyelitis and questions would often give other clues such as an overlying ulcer, which is not present in this case.



Next question >

## Charcot joint \*

A Charcot joint is also commonly referred to as a neuropathic joint. It describes a joint which has become badly disrupted and damaged secondary to a loss of sensation. In years gone by they were most commonly caused by neuropathy secondary to syphilis (tabes dorsalis) but are now most commonly seen in diabetics.

#### **Features**

- Charcot joints are typically a lot less painful than would be expected given the degree of joint disruption due to the sensory neuropathy. However, 75% of patients report some degree of pain
- the joint is typically swollen, red and warm
- as the condition progresses, the affected joint becomes unstable → abnormal movements and increased risk of fractures and dislocations
- progressive joint destruction can cause significant deformities
  - common deformities include a collapsed arch in the foot (commonly referred to as 'rocker-bottom' foot) or severe joint misalignment
- secondary complications such as skin ulceration and infection can occur due to repeated trauma and poor wound healing



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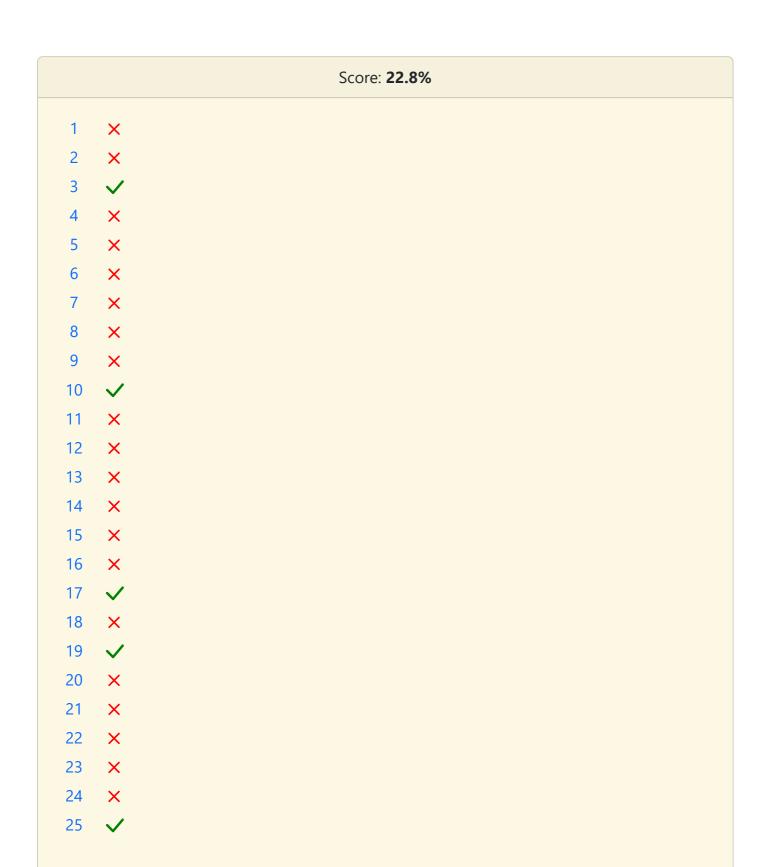
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Extensive bone remodeling / fragmentation involving the midfoot in this patient with a poorly controlled diabetes, compatible with Charcot's joint (neuropathic arthropathy)



Next question >

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Question 36 of 79





A 23-year-old woman is investigated for abdominal pain and haematuria. She started developing abdominal pain around 2 months ago and it is generally worse after eating. She also reports arthralgia, weight loss of 3kg and general lethargy.

On examination blood pressure is 150/102 mmHg, pulse 84/min and temperature 37.6°C. Dipstick examination of her urine shows blood+++.

## Bloods show the following:

Na <sup>+</sup>	142 mmol/l
K <sup>+</sup>	4.9 mmol/l
Urea	8.9 mmol/l
Creatinine	123 µmol/l
CRP	73 mg/l

An angiogram is performed:



# What is the most likely diagnosis?

Renal cell cancer	
O Polyarteritis nodosa	
Henoch-Schonlein purpura	×
Antiphospholipid syndrome	×

# Submit answer

Reference ranges  $\checkmark$ 

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Question 36 of 79



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A 23-year-old woman is investigated for abdominal pain and haematuria. She started developing abdominal pain around 2 months ago and it is generally worse after eating. She also reports arthralgia, weight loss of 3kg and general lethargy.

On examination blood pressure is 150/102 mmHg, pulse 84/min and temperature 37.6°C. Dipstick examination of her urine shows blood+++.

Bloods show the following:

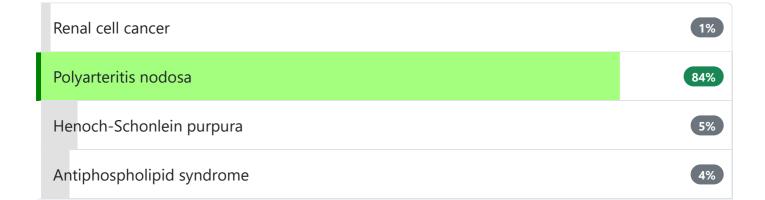
Na <sup>+</sup>	142 mmol/l
K <sup>+</sup>	4.9 mmol/l
Urea	8.9 mmol/l
Creatinine	123 µmol/l
CRP	73 mg/l

An angiogram is performed:



© Image used on license from Radiopaedia

# What is the most likely diagnosis?



The combination of systemic symptoms, fever, hypertension and renal failure are supportive of polyarteritis nodosa. The abdominal pain is due to the arteries supplying the gut being affected.

Both kidneys demonstrate beading and numerous microaneurysms affecting the intrarenal vessels. Similar changes are seen affecting the intrahepatic vessels with a few small microaneurysms noted. The proximal branches of the SMA appears normal; however there are no normal straight arteries from the jejunal arteries and lack of normal anastomotic arcades and loops. This is associated with multiple microaneurysms.



Next question >

# Polyarteritis nodosa 🖈

Polyarteritis nodosa (PAN) is a vasculitis affecting medium-sized arteries with necrotizing inflammation leading to aneurysm formation. PAN is more common in middle-aged men and is associated with hepatitis B infection.

#### **Features**

- fever, malaise, arthralgia
- weight loss
- hypertension
- mononeuritis multiplex, sensorimotor polyneuropathy
- testicular pain
- livedo reticularis
- haematuria, renal failure
- perinuclear-antineutrophil cytoplasmic antibodies (ANCA) are found in around 20% of patients with 'classic' PAN
- hepatitis B serology positive in 30% of patients



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## Livedo reticularis



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Angiogram from a patient with polyarteritis nodosa. Both kidneys demonstrate beading and numerous microaneurysms affecting the intrarenal vessels. Similar changes are seen affecting the intrahepatic vessels with a few small microaneurysms noted. The proximal branches of the SMA appears normal; however there are no normal straight arteries from the jejunal arteries and lack of normal anastomotic arcades and loops. This is associated with multiple microaneurysms.

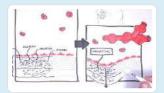


## **Textbooks**

High-yield textbook

Extended textbook

## Media



Polyarteritis Nodosa and Kawasaki Disease (Medium Vessel Vasculitis) - Symptoms, pathophysiology

Armando Hasudungan - YouTube

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Question 37 of 79





This 60-year-old woman who is being treated for heartburn comes for review. She has developed some spots on her lips:



## What is the most likely diagnosis?

CREST syndrome	
Oesophageal cancer	
Vitamin C deficiency	
Peutz-Jeghers syndrome	
Iron-deficiency anaemia	

Submit answer

Reference ranges  $\vee$ 

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Question 37 of 79



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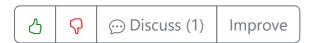
This 60-year-old woman who is being treated for heartburn comes for review. She has developed some spots on her lips:



What is the most likely diagnosis?



The heartburn may be explained by oesophageal dysmotility, a feature of CREST syndrome. The lesions on her lips are telangiectasia. She also has the typical tightening of the facial skin seen in patients with systemic sclerosis.



## Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

### Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

### Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - o patients with renal disease should be started on an ACE inhibitor
- poor prognosis

#### Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







#### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis

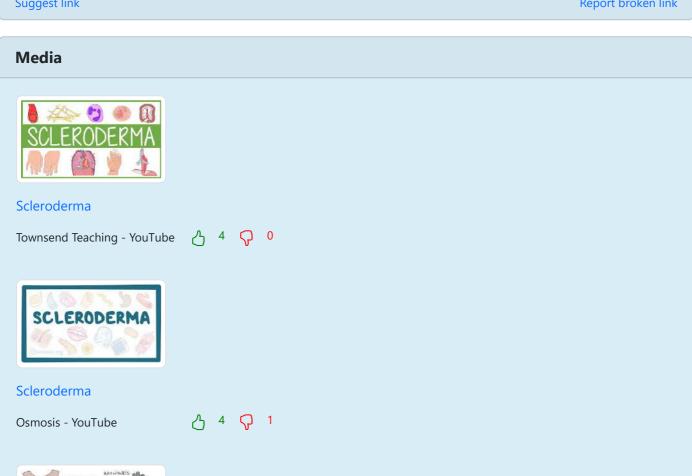


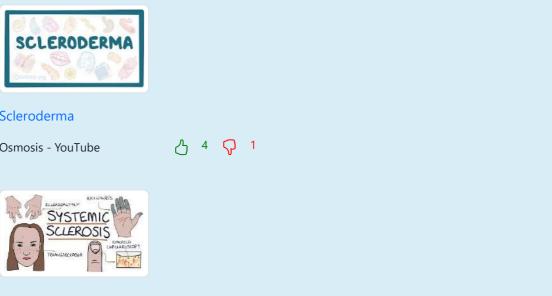












Zero to Finals - YouTube

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Question 38 of 79





A 52-year-old man presents with feeling generally unwell. For the past few weeks he has felt lethargic, lost weight and had troublesome testicular pain. Over the past week he has also noticed a 'weakness at the ankle' on the right side.

On examination blood pressure is 164/96 mmHg, pulse 86/min, temperature 37.3°C. Examination of the cardiovascular system is unremarkable. Foot drop is noted on the right side consistent with a common peroneal nerve palsy. A rash is also noted on his legs:



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A urine dipstick shows blood++. What is the most likely diagnosis?

Henoch-Schonlein purpura	
Wegener's granulomatosis	
Polyarteritis nodosa	
Amyloidosis	

# Submit answer

Reference ranges  $\checkmark$ 

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Question 38 of 79

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A urine dipstick shows blood++. What is the most likely diagnosis?



The correct answer is **Polyarteritis nodosa**. This patient presents with a combination of constitutional symptoms (lethargy and weight loss), testicular pain, hypertension, common peroneal nerve palsy (right-sided foot drop), and livedo reticularis on the legs. These findings are consistent with polyarteritis nodosa (PAN), a systemic necrotizing vasculitis that affects medium-sized arteries. PAN can cause multiorgan involvement, leading to a variety of clinical presentations. In this case, the patient's hypertension may be due to renal artery involvement, while the testicular pain could result from inflammation of the testicular artery. The common peroneal nerve palsy is likely secondary to ischemia caused by vasculitic changes in vasa nervorum.

**Henoch-Schonlein purpura** is an incorrect answer because this condition typically presents in children and is characterized by palpable purpura, arthritis or arthralgia, abdominal pain, and renal involvement. While there is some overlap with the presented symptoms, Henoch-Schonlein purpura would not explain the patient's testicular pain or common peroneal nerve palsy.

**Wegener's granulomatosis**, now known as granulomatosis with polyangiitis (GPA), is another form of vasculitis that primarily affects small- to medium-sized vessels. It classically presents with upper and lower respiratory tract symptoms and glomerulonephritis. Although it can potentially cause similar symptoms as PAN, GPA typically involves granulomatous inflammation which isn't mentioned in this scenario.

**Amyloidosis** is a group of diseases characterized by extracellular deposition of misfolded proteins called amyloid fibrils. While amyloidosis can cause multisystem involvement leading to diverse clinical manifestations such as nephrotic syndrome, cardiomyopathy, and peripheral neuropathy, the presence of livedo reticularis and testicular pain are not typical features of this disease.

**Behcet's syndrome** is a rare multisystem inflammatory disorder characterized by recurrent oral and genital ulcers, uveitis, and skin lesions. Although it can cause systemic vasculitis affecting both arteries and veins, the patient's presentation with hypertension, common peroneal nerve palsy, and testicular pain is less suggestive of Behcet's syndrome compared to polyarteritis nodosa.



Next question >

### Polyarteritis nodosa 🖈

Polyarteritis nodosa (PAN) is a vasculitis affecting medium-sized arteries with necrotizing inflammation leading to aneurysm formation. PAN is more common in middle-aged men and is associated with hepatitis B infection.

- fever, malaise, arthralgia
- weight loss
- hypertension
- mononeuritis multiplex, sensorimotor polyneuropathy
- testicular pain
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Livedo reticularis



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Angiogram from a patient with polyarteritis nodosa. Both kidneys demonstrate beading and numerous microaneurysms affecting the intrarenal vessels. Similar changes are seen affecting the intrahepatic vessels with a few small microaneurysms noted. The proximal branches of the SMA appears normal; however there are no normal straight arteries from the jejunal arteries and lack of normal anastomotic arcades and loops. This is associated with multiple microaneurysms.

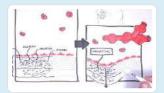


### **Textbooks**

High-yield textbook

Extended textbook

### Media



Polyarteritis Nodosa and Kawasaki Disease (Medium Vessel Vasculitis) - Symptoms, pathophysiology

Armando Hasudungan - YouTube



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A 8-year-old boy is complains of progressively worsening pain in both groin areas. He has no past medical history of note and his immunisations are up-to-date. There is no recent history of trauma. On examination he walks with a limp. An x-ray is requested:



### What is the most likely diagnosis?

Developmental dysplasia of the hip	
Slipped upper femoral epiphysis	
Osteosarcoma	
Acute lymphoblastic leukaemia	
Perthes disease	

Submit answer

### Score: 0%





Question 39 of 79



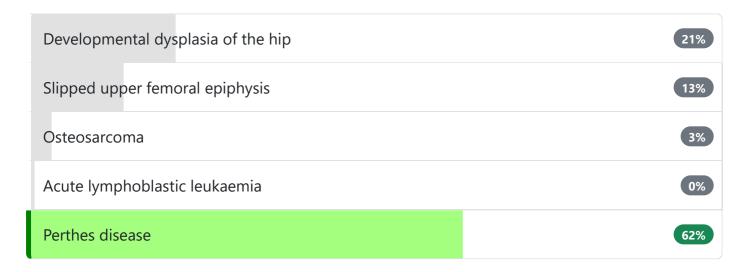




A 8-year-old boy is complains of progressively worsening pain in both groin areas. He has no past medical history of note and his immunisations are up-to-date. There is no recent history of trauma. On examination he walks with a limp. An x-ray is requested:



### What is the most likely diagnosis?



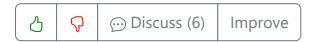
The correct answer is **Perthes disease**. Perthes disease, also known as Legg-Calve-Perthes disease, is a childhood condition that affects the hip joint. It occurs when blood supply to the femoral head is temporarily interrupted, leading to avascular necrosis of the bone. This results in pain and limping, which are common symptoms seen in this patient. The x-ray image provided shows characteristic changes of Perthes disease, with flattening and irregularity of the femoral head.

**Developmental dysplasia of the hip** is an incorrect option because it typically presents earlier in life and is associated with hip instability or dislocation rather than pain and limping. The x-ray findings in developmental dysplasia of the hip would show shallow acetabulum and potential dislocation or subluxation of the femoral head.

**Slipped upper femoral epiphysis** (SUFE) is also an incorrect choice. SUFE usually occurs during adolescence and presents with acute or chronic pain in the groin, thigh, or knee. The x-ray findings would demonstrate displacement of the metaphysis from the epiphysis along the growth plate. In this case, there is no evidence of such displacement on x-ray.

**Osteosarcoma** is a type of bone cancer that can cause pain and swelling around a joint but it's less likely to be the cause for this patient's symptoms given his age and lack of other signs like swelling or systemic symptoms. Additionally, osteosarcoma would present with lytic or sclerotic lesions on x-ray which are not evident in this case.

Finally, **acute lymphoblastic leukaemia** (ALL) can sometimes present with bone pain due to infiltration by leukaemic cells; however, ALL would be unlikely to cause localized bilateral groin pain without other systemic symptoms such as fever, fatigue, or weight loss. The x-ray findings in ALL would typically show diffuse osteopenia and metaphyseal bands, which are not present in this case.



Next question >

### Perthes' disease

Perthes' disease is a degenerative condition affecting the hip joints of children, typically between the ages of 4-8 years. It is due to avascular necrosis of the femoral head, specifically the femoral epiphysis. Impaired blood supply to the femoral head causes bone infarction.

Perthes' disease is 5 times more common in boys. Around 10% of cases are bilateral

### **Features**

- hip pain: develops progressively over a few weeks
- limp
- stiffness and reduced range of hip movement
- x-ray: early changes include widening of joint space, later changes include decreased femoral head size/flattening

### Diagnosis

plain x-ray

• technetium bone scan or magnetic resonance imaging if normal x-ray and symptoms persist

### Complications

- osteoarthritis
- premature fusion of the growth plates





### Catterall staging

Stage	Features
Stage 1	Clinical and histological features only
Stage 2	Sclerosis with or without cystic changes and preservation of the articular surface
Stage 3	Loss of structural integrity of the femoral head
Stage 4	Loss of acetabular integrity

### Management

- To keep the femoral head within the acetabulum: cast, braces
- If less than 6 years: observation
- Older: surgical management with moderate results
- Operate on severe deformities

### **Prognosis**

• Most cases will resolve with conservative management. Early diagnosis improves outcomes.

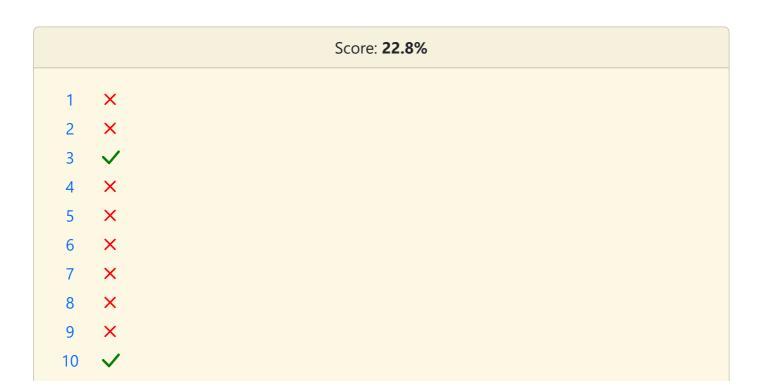


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## Textbooks High-yield textbook Extended textbook Media





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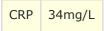


Question 40 of 79

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A 52-year-old female presents to rheumatology outpatient clinic with three months history of severe pain on her hands. She has no significant past medical history. On examination, there are swelling and erythema of the first, second and third metacarpophalangeal joints on both hands. She is diagnosed with rheumatoid arthritis.



What treatment should be started?

Methotrexate + infliximab	
Infliximab	
Methotrexate + prednisolone + sulfasalazine	
Methotrexate + prednisolone	
Azathioprine	

Submit answer

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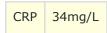


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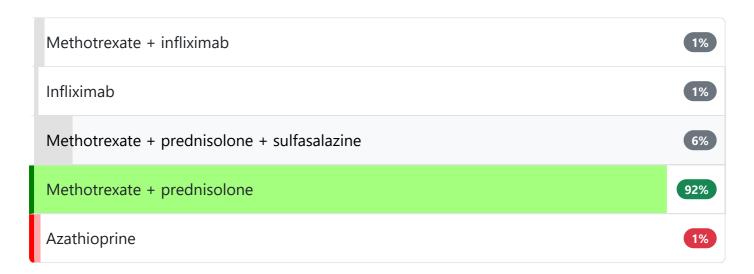


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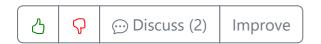
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What treatment should be started?



In 2018 NICE updated their rheumatoid arthritis guidelines. They now recommend disease-modifying antirheumatic drug (DMARD) monotherapy with a short-course of bridging prednisolone. In the past dual DMARD therapy was advocated as the initial step.



Next question >

### Rheumatoid arthritis: management \*

The management of rheumatoid arthritis (RA) has been revolutionised by the introduction of disease-modifying therapies in the past decade.

Patients with evidence of joint inflammation should start a combination of disease-modifying drugs (DMARD) as soon as possible. Other important treatment options include analgesia,

physiotherapy and surgery.

### Initial therapy

- NICE recommend DMARD **monotherapy** +/- a short-course of bridging prednisolone. In the past dual DMARD therapy was advocated as the initial step
- choices for initial DMARD monotherapy:
  - methotrexate is the most widely used DMARD. Monitoring of FBC & LFTs is essential
    due to the risk of myelosuppression and liver cirrhosis. Other important side-effects
    include pneumonitis
  - o sulfasalazine
  - o leflunomide
  - hydroxychloroquine: should only be considered for initial therapy if mild or palindromic disease

### Monitoring response to treatment

 NICE recommends using a combination of CRP and disease activity (using a composite score such as DAS28) to assess response to treatment

### Flares

• flares of RA are often managed with corticosteroids - oral or intramuscular

### **TNF-inhibitors**

- the current indication for a TNF-inhibitor is an inadequate response to at least two DMARDs including methotrexate
- etanercept: recombinant human protein, acts as a decoy receptor for TNF- $\alpha$ , subcutaneous administration, can cause demyelination, risks include reactivation of tuberculosis
- infliximab: monoclonal antibody, binds to TNF- $\alpha$  and prevents it from binding with TNF receptors, intravenous administration, risks include reactivation of tuberculosis
- adalimumab: monoclonal antibody, subcutaneous administration

### Rituximab

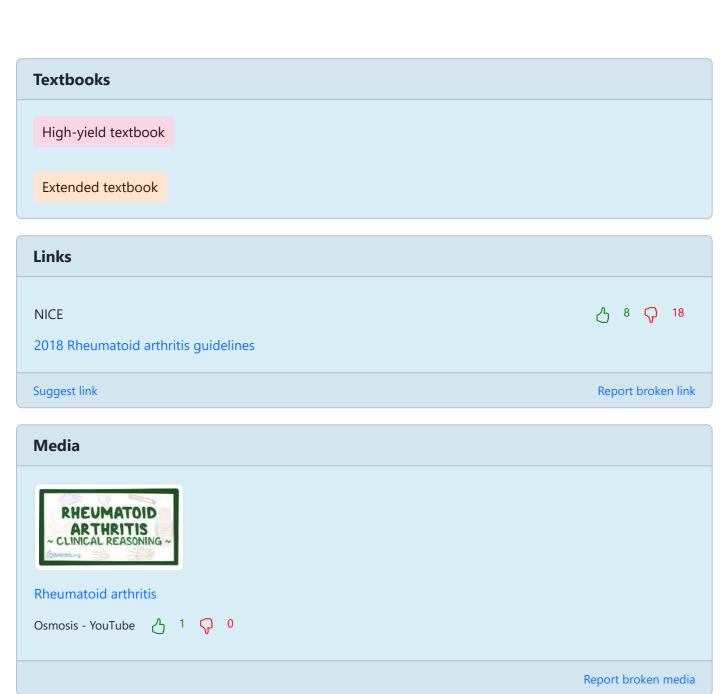
- anti-CD20 monoclonal antibody, results in B-cell depletion
- two 1g intravenous infusions are given two weeks apart
- infusion reactions are common

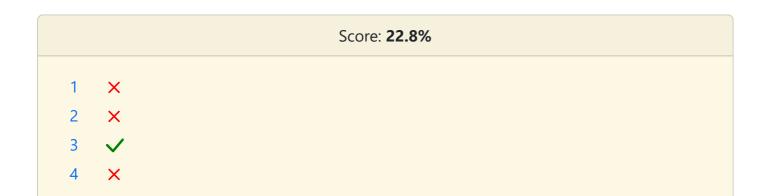
### **Abatacept**

- fusion protein that modulates a key signal required for activation of T lymphocytes
- leads to decreased T-cell proliferation and cytokine production
- given as an infusion
- not currently recommend by NICE









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A 52-year-old female has presented to your neurology clinic reporting difficulty in lifting her arms during exercises at the gym over the past two months. She is distressed by her symptoms and is tearful, mentioning that she is also using significant amounts of make-up to cover a new purple rash and swelling around her eyelids. She reports no past medical history no recent trauma and is normally fit and well. She has had a dry cough for the past 5 months that she puts down to her previous social smoking when she would smoke up to 2 cigarettes whilst going out with friends every 2 weeks.

On examination, you note a limited range of passive movement in both shoulders and hips secondary to tender deltoids and hip flexors. Examination of power demonstrates 4- out of 5 symmetrically in hip flexion and shoulder abduction. An elliptical erythematous rash is present around her eyes, the skin around her fingers appear tough bilaterally. Auscultation of her chest reveals bibasal fine inspiratory crackles and normal heart sounds. Observations show she is currently has a low-grade temperature of 37.7 degrees. A chest radiograph demonstrates bilateral fibrotic changes.

Her admission blood tests are as follows:

Hb	121 g/l
Platelets	590 * 10 <sup>9</sup> /l
WBC	12.3 * 10 <sup>9</sup> /l
ESR	20 mm/hr
Creatine kinase	3000 u/l
LDH	250 u/l

What is the most likely unifying diagnosis?

Inclusion body myositis	
Systemic sclerosis	
Dermatomyositis	
Polymyalgia rheumatica	
Fibromyalgia	

Submit answer

### Score: 0%

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A 52-year-old female has presented to your neurology clinic reporting difficulty in lifting her arms during exercises at the gym over the past two months. She is distressed by her symptoms and is tearful, mentioning that she is also using significant amounts of make-up to cover a new purple rash and swelling around her eyelids. She reports no past medical history no recent trauma and is normally fit and well. She has had a dry cough for the past 5 months that she puts down to her previous social smoking when she would smoke up to 2 cigarettes whilst going out with friends every 2 weeks.

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WBC	12.3 * 10 <sup>9</sup> /l
ESR	20 mm/hr
Creatine kinase	3000 u/l
LDH	250 u/l

What is the most likely unifying diagnosis?



The patient has presented with a syndrome of proximal myopathy associated with a violaceous

rash on her eyelid and mechanics hands, in addition to possible interstitial lung disease. The most suspicious diagnosis is an inflammatory myositis. Inclusion body myositis is a diagnosis of exclusion and is normally isolated to proximal and less commonly distal, bulbar and facial muscles. Systemic sclerosis is a possible diagnosis and can produce inflammatory myositis similar to polymyositis. However, cranial and peripheral neuropathies are more common. The distinctive cutaneous features in this patient are strongly suggestive of dermatomyositis: the violaceous eyelid rash and oedema is a heliotropic rash associated with dermatomyositis. Other cutaneous findings include Gottron's papules (violaceous papules on the extensor surfaces of fingers), shawl and V sign (photosensitive hyperpigmentation around the shoulders and upper chest), mechanics hands and periungual erythema. Note the normal ESR result, which is often not elevated in dermatomyositis patients.



Next question >

### Dermatomyositis \*

### Overview

- an inflammatory disorder causing symmetrical, proximal muscle weakness and characteristic skin lesions
- may be idiopathic or associated with connective tissue disorders or underlying malignancy (typically ovarian, breast and lung cancer, found in 20-25% more if patient older). Screening for an underlying malignancy is usually performed following a diagnosis of dermatomyositis
- polymyositis is a variant of the disease where skin manifestations are not prominent

### Skin features

- photosensitive
- macular rash over back and shoulder
- heliotrope rash in the periorbital region
- Gottron's papules roughened red papules over extensor surfaces of fingers
- 'mechanic's hands': extremely dry and scaly hands with linear 'cracks' on the palmar and lateral aspects of the fingers
- nail fold capillary dilatation

### Other features

- proximal muscle weakness +/- tenderness
- Raynaud's
- respiratory muscle weakness
- interstitial lung disease: e.g. Fibrosing alveolitis or organising pneumonia
- dysphagia, dysphonia

### Investigations

- the majority of patients (around 80%) are ANA positive
- around 30% of patients have antibodies to aminoacyl-tRNA synthetases (anti-synthetase antibodies), including:
  - o antibodies against histidine-tRNA ligase (also called Jo-1)
  - o antibodies to signal recognition particle (SRP)
  - o anti-Mi-2 antibodies



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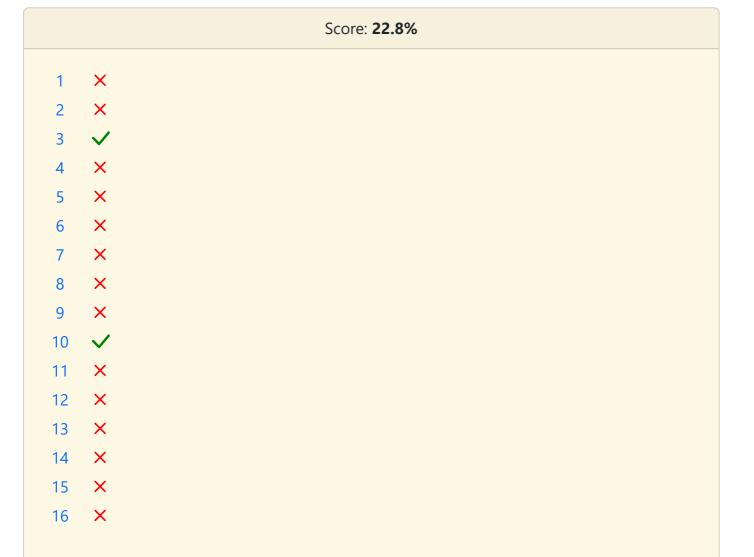




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### Question 42 of 79

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A 63-year-old female presents to gastroenterology outpatient clinic with a four-week history of gastric reflux, which has not improved despite being prescribed both ranitidine and omeprazole by her GP. She is awaiting an urgent OGD to investigate symptoms further. She reports having lost 7kg in weight over the past 6 months and is also distressed by appearances of white hard lumps appearing on her fingertips. On examination, you note cool peripheries and dry mucous membranes, left thumb calcinosis surrounded by shiny skin up to her wrist joint and wrinkling of skin around her mouth. Her blood tests are as follows demonstrate she is positive for anticentromere antibodies. What is the most likely diagnosis?

Diffuse cutaneous systemic sclerosis	
Systemic sclerosis sine scleroderma	
Zollinger-Ellison syndrome	
Limited cutaneous systemic sclerosis	
Raynaud's syndrome	

Submit answer

Reference ranges  $\vee$ 

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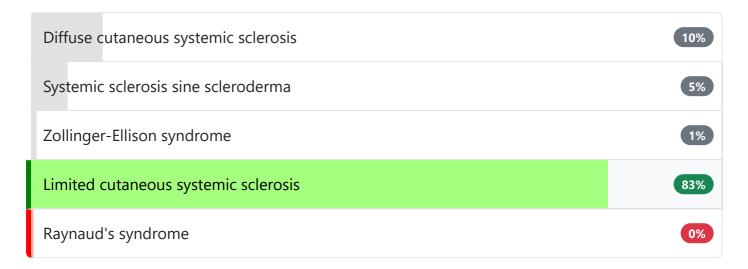
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Question 42 of 79 X

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Systemic sclerosis (SS) is a fairly clear answer when presented with a combination of sclerodactyly, calcinosis, perioral puckering and gastro-oesophageal reflux symptoms in a middle-aged female. The main differentials are whether this represents diffuse cutaneous, limited cutaneous or systemic sclerosis sine scleroderma. The latter describes patients with systemic involvement and possible Raynaud's phenomenon in the absence of other cutaneous manifestations with detection of SS autoantibodies. SS can be described generally as diffuse when skin proximal to the distal forearm is involved, such as the elbow, thorax or abdomen. Note that both limited and diffuse cutaneous SS has extracutaneous involvement: however, patients with diffuse cutaneous SS are more likely to develop significant renal, lung and cardiac disease.

Autoantibodies are useful in confirming the subtype of SS and predict extracutaneous involvement but negative results do not rule out SS. Anti-centromere antibodies are associated with limited cutaneous SS1, anti-Scl 70 with diffuse SS and lung involvement<sup>1</sup>, anti-RNA polymerase III to those at high risk of scleroderma renal crisis<sup>2</sup>, anti-U3-RNP to those at high risk of pulmonary hypertension<sup>3</sup> and anti-PM-Scl to those at high risk of SS associated myositis<sup>4</sup>.

- 1. Reveille JD, Solomon DH et al. Evidence-based guidelines for the use of immunologic tests: anticentromere, ScI-70, and nucleolar antibodies. Arthritis Rheum. 2003;49(3):399
- 2. Nguyen B, Mayes MD, Arnett FC et al. HLA-DRB1\*0407 and \*1304 are risk factors for scleroderma renal crisis. Arthritis Rheum. 2011;63(2):530.

- 3. Sacks DG, Okano Y, Steen VD et al. Isolated pulmonary hypertension in systemic sclerosis with diffuse cutaneous involvement: association with serum anti-U3RNP antibody. J Rheumatol. 1996;23(4):639
- 4. Oddis CV, Okano Y, Rudert WA et al. Serum autoantibody to the nucleolar antigen PM-Scl. Clinical and immunogenetic associations. Arthritis Rheum. 1992;35(10):1211



Next question >

# Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - patients with renal disease should be started on an ACE inhibitor
- poor prognosis

Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







# **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis

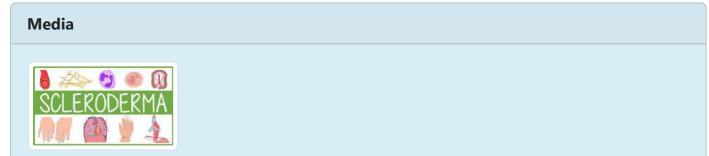


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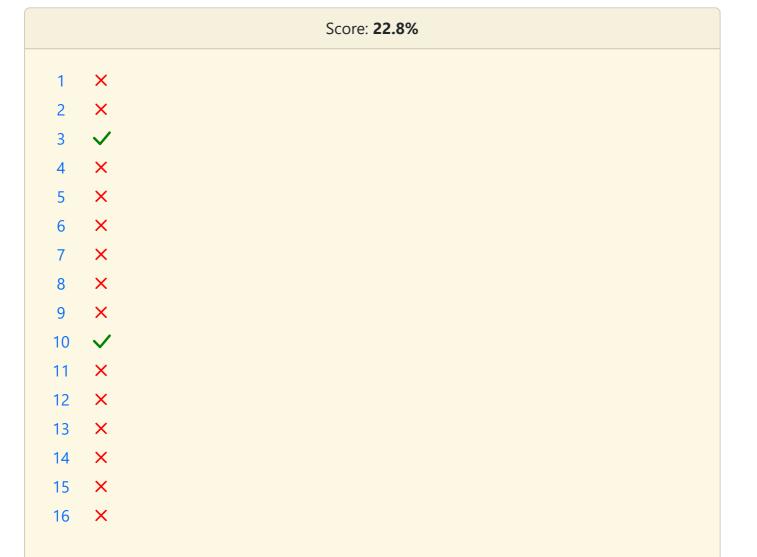








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### Question 43 of 79

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A 27-year-old male presents after recently returning from Bangladesh with 2 weeks of daily spiking fever, a new rash on his foot and pain on bending his knees or closing his hands. He also reports lumps and bumps on his neck that he thinks are new. He denies any cough or weight loss. He has no other past medical history and is unaware of any unwell family members. On examination, his temperature is 39.2 degrees. You note a maculopapular rash on his left sole and face. His knees and wrists are swollen and tender. His chest and cardiovascular examination are unremarkable, his abdomen is soft. However, you note a 12cm splenomegaly. His serum tests demonstrate:

Hb	127 g/l
Platelets	450 * 10 <sup>9</sup> /l
WBC	17.0 * 10 <sup>9</sup> /l
Neuts	11.0 * 10 <sup>9</sup> /l

Na <sup>+</sup>	138 mmol/l
K <sup>+</sup>	3.5 mmol/l
Urea	7.8 mmol/l
Creatinine	70 µmol/l

CRP	30 mg/l
Ferritin	2000 μg/l
ALP	250 u/l
ALT	160 u/l
ANA	negative
dsDNA	negative

His chest radiograph appears unremarkable with no focal consolidation. A first induced sputum is negative for acid-fast bacilli. What is the most likely diagnosis?

Miliary tuberculosis	
Adult onset Stills disease	
Reactive arthritis post-travellers diarrhoea	

Porphyria cutanea tarda	
Systemic lupus erythematosus	

# Submit answer

Reference ranges ✓

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Question 43 of 79

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ALT	160 u/l
ANA	negative
dsDNA	negative

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# Systemic lupus erythematosus

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Adult onset Stills disease (ASD) is a systemic inflammatory condition of unknown aetiology but often thought to be secondary to an infectious trigger on a background of genetic predisposition. It classically presents in young adults under 40 years old, peaking between 15-25 years, with a daily fever and a new non-pruritic rash. The most sensitive diagnostic classification is the Yamaguchi criteria<sup>1</sup>. defined by a patient presenting with all major criteria and at least two minor criteria.

## Major criteria:

- Fever greater than 39 degrees over one week
- Arthralgia or arthritis lasting two weeks or longer
- Non-pruritic or maculopapular rash, salmon coloured, found on trunk or extremities, particularly during febrile episodes
- Leucocytosis > 10,000/microL

### Minor criteria:

• Sore throat, lymphadenopathy, hepatomegaly or splenomegaly, abnormal liver function tests, negative ANA or rheumatoid factor

A number of features in the history and examination should point you towards ASD. The nature of the fever is quotidian: ASD fevers tend to spike daily or twice daily. Secondly, the location of the rash is also characteristic, most commonly a salmon coloured rash in the trunk or the soles, palms and face. Knees, wrist and ankles are the most common sites for arthritis and arthralgias. Ferritin rises are characteristic of ASD, observed in up to 70% of patients<sup>2</sup>. Mild elevations in ALT and ALP are also characteristic of ASD. However, the patient does not describe a sore throat, which is often severe and non-suppurative in up to 70% of ASD patients. Treatment of ASD involves systemic immunosuppression, initially with prednisolone followed by DMARDs.

The main differential to consider is TB. A two-week history is short for miliary TB and particularly unusual for someone without chest signs. It typically is the result of a pulmonary focus eroding the pulmonary vein, leading to systemic dissemination. A negative induced sputum also points against miliary Tb but does not fully exclude it. SLE could also fit with the clinical picture but again, less likely with negative dsDNA and ANA. There is no suggestion that the patient has recently had GI symptoms. Reactive arthritis, formerly known as Reiter's syndrome, does not account for the splenomegaly or serum abnormalities.

- 1. Yamaguchi M, Ohta A, Tsunematsu T et al. Preliminary criteria for classification of adult Still's disease. J Rheumatol. 1992;19(3):424
- 2. Ohta A, Yamaguchi M, Tsunematsu T et al. Adult Still's disease: a multicenter survey of Japanese patients. J Rheumatol. 1990;17(8):1058

Next question >

# Still's disease in adults \*

# **Epidemiology**

• has a bimodal age distribution - 15-25 yrs and 35-46 yrs

### **Features**

S

- arthralgia
- elevated serum ferritin
- rash: salmon-pink, maculopapular
- pyrexia
  - typically rises in the late afternoon/early evening in a daily pattern and accompanies a worsening of joint symptoms and rash
- lymphadenopathy
- rheumatoid factor (RF) and anti-nuclear antibody (ANA) negative

The diagnosis of Still's disease in adults can be challenging. The Yamaguchi criteria is the most widely used criteria and has a sensitivity of 93.5%.

# Management

- NSAIDs
  - should be used first-line to manage fever, joint pain and serositis
  - o they should be trialled for at least a week before steroids are added.
- steroids
  - may control symptoms but won't improve prognosis
- if symptoms persist, the use of methotrexate, IL-1 or anti-TNF therapy can be considered



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An 88-year-old female is admitted to hospital after recurrent mechanical falls. Her past medical history includes an anterior resection for sigmoid carcinoma, type 2 diabetes mellitus and gout.

Three days into her admission, she was treated for hospital-acquired pneumonia with three days of intravenous tazocin. One week into her admission, she developed a swollen inflamed 2nd MTP joint and colchicine was started.

After becoming medically stable 10 days into admission and awaiting a package of care at home, nursing staff report diarrhoea, with type 7 stool up to 7 times a day. She has no laxatives prescribed. One set of stool cultures were sent within 15 minutes of the last episode, which have proved negative for *Clostridium difficile* toxin and, MC+S and norovirus.

What is the most likely cause of her diarrhoea?

Clostridium difficile	
Norovirus	
Colchicine	
Recurrence of colon carcinoma	
Tazocin	

Submit answer

Reference ranges  $\vee$ 

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Question 44 of 79



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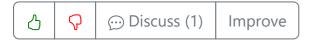
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What is the most likely cause of her diarrhoea?



Colchicine is a classic cause of diarrhoea, particularly in the elderly and in higher doses, leading to the old adage of 'you run before you walk!' Antibiotics can induce diarrhoea without infection, particularly those acting on anaerobes<sup>1</sup>. It is unusual in this timeframe, having diarrhoea 4 days after the antibiotics finished. However, the most serious cause in this scenario is whether tazocin has resulted in pseudomembranous colitis, resulting in *Clostridium difficile* infection. In this case, negative samples for antigens and toxins are likely to be a true negative result. The main concern regards samples that were not transported to the lab promptly (within 2 hours), resulting in the breakdown of *Clostridium difficile* toxin, hence a false negative result. There is little to suggest cancer recurrence or an outbreak of norovirus.

1. Barbut F, Meynard L. Managing antibiotic associated diarrhoea. BMJ 2002; 324



# Gout: management \*

Gout is a form of microcrystal synovitis caused by the deposition of monosodium urate monohydrate in the synovium. It is caused by chronic hyperuricaemia (uric acid > 450 µmol/l)

# Acute management

- NSAIDs or colchicine are first-line
  - the maximum dose of NSAID should be prescribed until 1-2 days after the symptoms have settled
  - o gastroprotection (e.g. a proton pump inhibitor) may also be indicated
- colchicine
  - inhibits microtubule polymerization by binding to tubulin, interfering with mitosis. Also inhibits neutrophil motility and activity
  - has a slower onset of action
  - may be used with caution in renal impairment: the BNF advises to reduce the dose if eGFR is 10-50 ml/min and to avoid if eGFR < 10 ml/min (BNF)
  - o the main side-effect is diarrhoea
- oral steroids may be considered if NSAIDs and colchicine are contraindicated.
  - o a dose of prednisolone 15mg/day is usually used
- another option is intra-articular steroid injection
- if the patient is already taking allopurinol it should be continued

# Indications for urate-lowering therapy (ULT)

- the British Society of Rheumatology Guidelines now advocate offering urate-lowering therapy to all patients after their **first attack of gout**
- ULT is particularly recommended if:
  - > = 2 attacks in 12 months
  - o tophi
  - o renal disease
  - uric acid renal stones
  - prophylaxis if on cytotoxics or diuretics

# **Urate-lowering therapy**

- it has traditionally been taught that urate-lowering therapy should not be started until 2 weeks after an acute attack, as starting too early may precipitate a further attack. The evidence base to support this however looks weak
- in 2017 the BSR updated their guidelines. They still support a delay in starting urate-lowering therapy because it is better for a patient to make long-term drug decisions whilst not in pain
  - the key passage is: 'Commencement of ULT is best delayed until inflammation has settled as ULT is better discussed when the patient is not in pain'
- allopurinol is first-line

- $\circ$  initial dose of 100 mg od, with the dose titrated every few weeks to aim for a serum uric acid of < 360  $\mu$ mol/l
- o a lower target uric acid level below 300 μmol/L may be considered for patients who have tophi, chronic gouty arthritis or continue to have ongoing frequent flares despite having a uric acid below 360 μmol/L
- o a lower initial dose of allopurinol should be given if the patient has a reduced eGFR
- colchicine cover should be considered when starting allopurinol. NSAIDs can be used if colchicine cannot be tolerated. The BSR guidelines suggest this may need to be continued for 6 months
- the second-line agent when allopurinol is not tolerated or ineffective is febuxostat (also a xanthine oxidase inhibitor)
- in refractory cases other agents may be tried:
  - uricase (urate oxidase) is an enzyme that catalyzes the conversion of urate to the degradation product allantoin. It is present in certain mammals but not humans
  - in patients who have persistent symptomatic and severe gout despite the adequate use
    of urate-lowering therapy, pegloticase (polyethylene glycol modified mammalian uricase)
    can achieve rapid control of hyperuricemia. It is given as an infusion once every two
    weeks

# Lifestyle modifications

- reduce alcohol intake and avoid during an acute attack
- lose weight if obese
- avoid food high in purines e.g. Liver, kidneys, seafood, oily fish (mackerel, sardines) and yeast products

# Other points

- consideration should be given to stopping precipitating drugs (such as thiazides)
- losartan has a specific uricosuric action and may be particularly suitable for the many patients who have coexistent hypertension
- increased vitamin C intake (either supplements or through normal diet) may also decrease serum uric acid levels

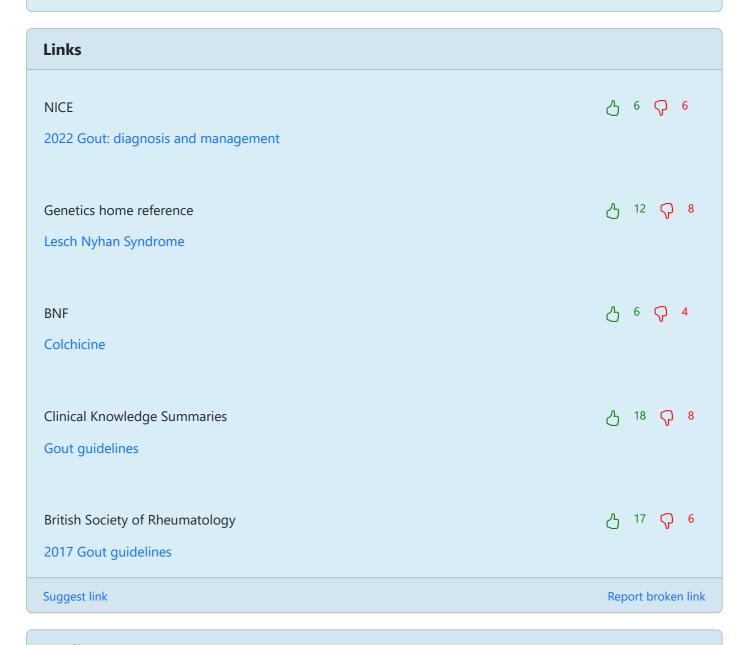


Next question >



High-yield textbook

Extended textbook







### Gout

Rhesus Medicine - YouTube

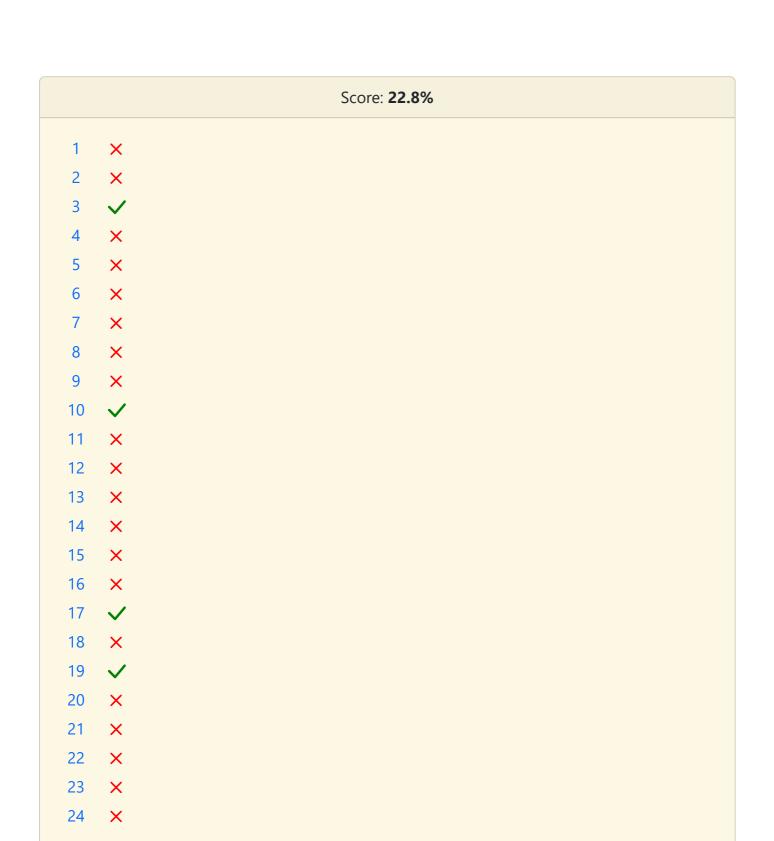






Gout





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Question 45 of 79

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A 28-year-old woman who is 20 weeks pregnant is referred to you by her GP. She has a 2-month history of arthralgia, myalgia, and fatigue. She had initially put this down to pregnancy but was finding it increasingly difficult to do her job as a health care assistant in a local nursing home. She denied any shortness of breath, swallowing difficulties or alopecia.

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On examination there was tenderness of the 2nd and 3rd metacarpophalangeal (MCP) joints bilaterally and both wrists but no evidence of active synovitis. There are several painless mouth ulcers. You notice a few bruises on her arms but no other evidence of a rash. Her chest was clear and heart sounds were normal. Neurological examination was normal including full visual fields and eye movements.

# Her bloods showed the following:

Haemoglobin	108 g/L
White Cell Count	9.2 x 10 <sup>9</sup> /L
Platelets	103 x 10 <sup>9</sup> /L
Neutrophils	6.02 x 10 <sup>9</sup> /L
Lymphocytes	0.80 x 10 <sup>9</sup> /L
Eosinophils	0.90 x 10 <sup>9</sup> /L
ESR	29 mm/h

Urea	6.9 mmol/L
Creatinine	118 micromol/L
CRP	11 mg/L
Alkaline Phosphatase	87 iu/L

ALT	42 iu/L
Albumin	32 g/L

ANA	1: 320
dsDNA	24
Anti -Ro	Positive
Anti -La	Positive
Rheumatoid Factor	Positive
Anti CCP	Negative
Antiphospholipid antibody	negative

Given the most likely diagnosis, what complication needs to be discussed with her?

Post partum haemorrhage	
Congenital heart block	
Deep vein thrombosis	
Pre-eclampsia	
Scleritis	

Submit answer

Reference ranges  $\vee$ 

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Question 45 of 79



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ALT	42 iu/L
Albumin	32 g/L

ANA	1: 320
dsDNA	24
Anti -Ro	Positive
Anti -La	Positive
Rheumatoid Factor	Positive
Anti CCP	Negative
Antiphospholipid antibody	negative

Given the most likely diagnosis, what complication needs to be discussed with her?

Post partum haemorrhage	2%
Congenital heart block	83%
Deep vein thrombosis	8%
Pre-eclampsia	5%
Scleritis	3%

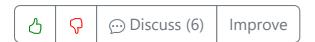
This question tests your knowledge of the diagnosis of Systemic Lupus Erythematosus (SLE). Diagnosis is based on the American College of Rheumatology (ACR) criteria written in 1982 and revised in 1997:

Four or more of the 11 criteria need to be fulfilled to be able to diagnose SLE. Note that while fatigue is a common feature it is not used in the diagnostic criteria. In this case, the criteria are oral ulcers + arthritis + positive dsDNA + the presence of ANA (while there is lymphopenia this is a single test result only)

In this case, there are sicca symptoms and Anti-Ro and -La antibodies suggesting an overlap with Sjogren's syndrome. Rheumatoid factor is positive in approximately 40% of SLE patients. The absence of anti-CCP should point you away from rheumatoid arthritis.

The point to make with this question is to test the candidate's knowledge of diagnosis of connective tissue diseases and their associated complications. Anti-Ro antibodies can cross the placenta and can lead to neonatal lupus and congenital heart block of the newborn, which can require pacing at birth. Miscarriage is another common complication of SLE. These can occur beyond the first trimester.

While postpartum haemorrhage, pre-eclampsia and deep vein thrombosis are complications of pregnancy that will need to be discussed, the presence of SLE does not increase the risk of either in this scenario. If the antiphospholipid antibody or lupus anticoagulant were positive then there is an increased risk of arterial or venous thrombosis, in which case you might consider anticoagulation but obviously not with warfarin in pregnancy.



Next question >

# Systemic lupus erythematosus: pregnancy

### Overview

- risk of maternal autoantibodies crossing the placenta
- leads to a condition termed neonatal lupus erythematosus
- neonatal complications include congenital heart block
- strongly associated withanti-Ro (SSA) antibodies



Next question >







American College of Rheumatology



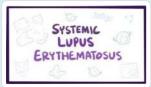




Systemic Lupus Erythematosus diagnostic criteria

Suggest link Report broken link

# Media



Systemic lupus erythematosus (SLE) - causes, symptoms, diagnosis & pathology

Osmosis - YouTube











Systemic Lupus Erythematosus (SLE) - signs and symptoms, pathophysiology, investigations, treatment

Armando Hasudungan - YouTube







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Question 46 of 79





You are the medical doctor on an acute medical admissions unit. A 56-year old female with hypertension, pulmonary fibrosis and a recent diagnosis of Raynaud's phenomenon presents with generally feeling unwell. On further questioning she also reports dysphagia for the past few months for which she is awaiting investigations under the gastroenterology team at your hospital. She is currently only on amlodipine 5mg od.

Her observations are: temperature 36.4°C, pulse 88/min, blood pressure 172/88 mmHg, respiratory rate 14/min, sats 100% on room air. Her chest is clear and abdomen soft, non-tender. Blood tests reveal an acute kidney injury with: sodium 141 mmol/l, potassium 4.6 mmol/l, urea 27 mmol/l, creatinine 320  $\mu$ mol/l (her GP notes state she had a normal renal function from a routine blood test 1 month ago).

What is the most appropriate treatment at this stage?

Fluids	
Stat 5mg amlodipine	
Stat angiotensin-converting enzyme inhibitor (ACE-i)	
Haemodialysis	
Haemofiltration	

Submit answer

Reference ranges ∨

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Question 46 of 79



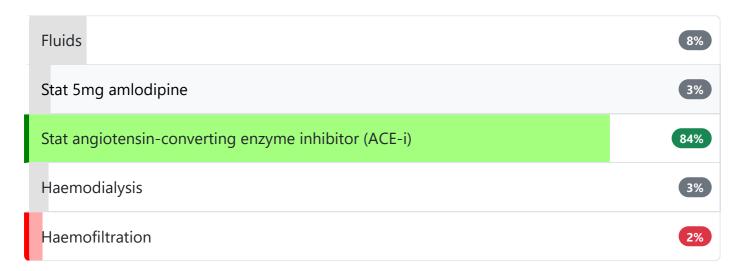
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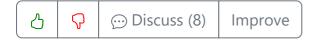
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What is the most appropriate treatment at this stage?



This lady has features of diffuse cutaneous systemic sclerosis - she has Raynaud's phenomenon, pulmonary fibrosis and dysphagia. Her current presentation is that of scleroderma renal crisis. This is a medical emergency and treatment should be administered as soon as possible. The most appropriate treatment initially would be an ACE-i with a consideration for dialysis and renal transplantation if required.



Next question >

#### Systemic sclerosis \*

Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

#### Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - patients with renal disease should be started on an ACE inhibitor
- poor prognosis

#### Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







#### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - o associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis



Next question >



## **Textbooks** High-yield textbook Extended textbook Links DermNet NZ Systemic sclerosis Suggest link Report broken link Media Scleroderma Townsend Teaching - YouTube 4 Q 0 SCLERODERMA Scleroderma ტ 4 ♀ 1 Osmosis - YouTube SYSTEMIC SCLEROSIS Systemic Sclerosis and Scleroderma Zero to Finals - YouTube Report broken media

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Question 47 of 79





A 68-year-old lady attends for review in the oncology clinic. She has advanced oestrogen receptor positive, HER2 negative breast cancer, with metastasis to her ribs, thoracic vertebrae and right humerus. She previously underwent right mastectomy and first line chemotherapy but has declined further chemotherapy.

She has had back and rib pain which was was improved by external beam radiotherapy. She was started on alendronate to help prevent pathological fractures but has since suffered nausea, severe acid reflux and epigastric discomfort not helped by a proton pump inhibitor. Her alendronate was stopped and risedronate was trialled but resulted insimilar effects and so was also discontinued.

What is the most appropriate medication to prescribe this lady to help prevent skeletal related events?

Denosumab	
Lapatinib	
Letrozole	
Strontium	
Trastuzumab	

Submit answer

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Question 47 of 79



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What is the most appropriate medication to prescribe this lady to help prevent skeletal related events?



This lady has bone metastases so is at risk of pathological fracture. The first choice agent would be a bisphosphonate such as alendronate. However, this lady is unable to take bisphosphonates due to oesophageal irritation and nausea are common side effects.

Denosumab is a monoclonal antibody which binds to and inhibits RANKL on the surface of osteoclasts, preventing the break down of bone. It is approved by NICE for women with osteoporosis or with advanced breast cancer and bone metastases, who have been intolerant to bisphosphonates.

Strontium ranelate is recommended for primary prevention of osteoporotic fractures in at risk women who have been intolerant of bisohosphonates. However, it has not been shown to reduce skeletal related events in bone metastases.

Lapatinib is a tyrosine kinase inhibitor of the HER2 and epidermal growth factor receptors and is part therapy to treat HER2 positive breast cancer.

Letrozole is an aromatise inhibitor used to treat oestrogen receptor positive breast cancer. Although it is generally effective against these types of cancer, it has not been shown in trials to reduce skeletal related events in patients with this cancer type.

Trastuzumab is a monoclonal antibody against the HER2 receptor and is part of therapy to treat HER2 positive breast cancer.

#### References:

National Institute for Health and Care Excellence. Denosumab for the prevention of skeletal-related events in adults with bone metastases from solid tumours. NICE technology appraisal guidance [TA265] (2012)

National Institute for Health and Care Excellence. Alendronate, etidronate, risedronate, raloxifene and strontium ranelate for the primary prevention of osteoporotic fragility fractures in postmenopausal women. NICE technology appraisal guidance 160 (2011)



Next question >

#### Denosumab \*

Denosumab is a relatively new treatment for osteoporosis. It is a human monoclonal antibody that prevents the development of osteoclasts by inhibiting RANKL. Remember that osteoblasts build bone, osteoclasts eat bone. It is given as a subcutaneous injection, at a dose of 60mg, every 6 months.

A larger dose of denosumab (120mg) may also be given every 4 weeks for the prevention of skeletal-related events (i.e. pathological fractures) in adults with bone metastases from solid tumours. For example, you may have noticed some of your breast cancer patients have been prescribed denosumab.

#### Where does it fit in the management of osteoporosis?

Oral bisphosphonates are still given first-line, with oral alendronate being the first-line treatment. If alendronate is not tolerated then NICE recommend using an alternative bisphosphonate - either risedronate or etidronate. Following this the advice becomes more complicated with the next-line medications only being started if certain T score and other risk factor criteria being met. Raloxifene and strontium ranelate were recommended as next-line drugs in the NICE criteria but following recent safety concerns regarding strontium ranelate it is likely there will be an increasing role for denosumab.

NICE published a technology appraisal looking at the role of denosumab in 2010. A link is

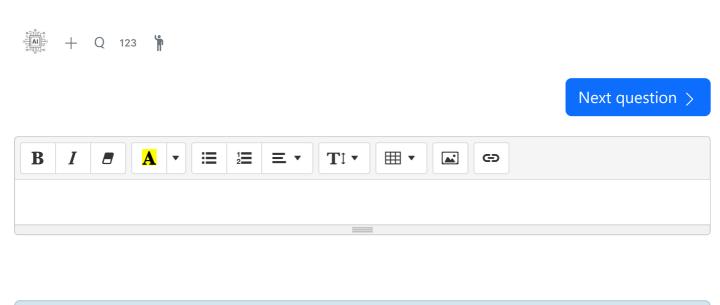
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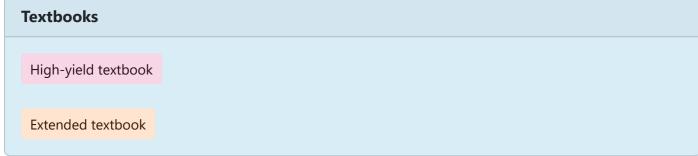
#### What are the known side-effects of denosumab?

Denosumab is generally well tolerated. Dyspnoea and diarrhoea are generally considered the two most common side effects, occuring in around 1 in 10 patients. Other less common side effects include hypocalcaemia and upper respiratory tract infections.

#### What does the Drug Safety Update add?

Cases of atypical femoral fractures have been noted in patients taking denosumab. Doctors are advised to look out for patients complaining of unusual thigh, hip or groin pain.







#### Media



#### Osteoporosis pharmacology, prevention and treatment

Armando Hasudungan - YouTube





Report broken media

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Question 48 of 79

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A 38-year-old male plumber is referred to the medical assessment unit by his GP due to reduced oxygen saturations. He has had mild asthma since childhood but no other medical history of note. His medications are a salbutamol inhaler when required and co-codamol for long standing back pain. On examination he is found to have an early diastolic murmur but no other abnormalities are detected. He goes on to have a chest x-ray which demonstrates apical interstitial shadowing. He undergoes pulmonary function tests which are as follows:

FEV1	1.9L	(Predicted 2.1-3.1)
FVC	2.2	(Predicted 3.0-4.4)
TLC	4.5	(Predicted 5.0-7.5)
Transfer factor (DLCO)	Low	

What is the most likely diagnosis?

Ankylosing spondylitis	
Asbestosis	
Extrinsic allergic alveolitis	
Churg-Strauss syndrome	
Sarcoidosis	

Submit answer

Reference ranges  $\vee$ 

## Score: **0**% 1 2 3 -

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Back to top





Question 48 of 79







A 38-year-old male plumber is referred to the medical assessment unit by his GP due to reduced oxygen saturations. He has had mild asthma since childhood but no other medical history of note. His medications are a salbutamol inhaler when required and co-codamol for long standing back pain. On examination he is found to have an early diastolic murmur but no other abnormalities are detected. He goes on to have a chest x-ray which demonstrates apical interstitial shadowing. He undergoes pulmonary function tests which are as follows:

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TLC	4.5	(Predicted 5.0-7.5)
Transfer factor (DLCO)	Low	

What is the most likely diagnosis?



There are a couple of red-herrings in this question designed to lead candidates astray. The fact that he is a plumber could be linked to a diagnosis of asbestosis, whilst his prior history of asthma may be suggestive of Churg-Strauss syndrome. However the fact that this patient's CXR shows apical interstitial shadowing, combined with the restrictive pattern of his pulmonary function tests indicate that he is suffering with apical lung fibrosis. This narrows the potential answers down to ankylosing spondylitis, sarcoidosis or extrinsic allergic alveolitis. The history of back pain and the finding of an early diastolic murmur (suggestive of aortic regurgitation) confirm ankylosing spondylitis as the most likely diagnosis.



#### Ankylosing spondylitis: features \*

Ankylosing spondylitis is a HLA-B27 associated spondyloarthropathy. It typically presents in males (sex ratio 3:1) aged 20-30 years old.

#### **Features**

- typically a young man who presents with lower back pain and stiffness of insidious onset
- stiffness is usually worse in the morning and improves with exercise
- the patient may experience pain at night which improves on getting up

#### Clinical examination

- reduced lateral flexion
- reduced forward flexion Schober's test a line is drawn 10 cm above and 5 cm below the back dimples (dimples of Venus). The distance between the two lines should increase by more than 5 cm when the patient bends as far forward as possible
- reduced chest expansion

#### Other features - the 'A's

- Apical fibrosis
- Anterior uveitis
- Aortic regurgitation
- Achilles tendonitis
- AV node block
- Amyloidosis
- and cauda equina syndrome
- peripheral arthritis (25%, more common if female)



Next question >

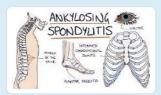


#### **Textbooks**

High-yield textbook

### Links NICE 2017 Spondyloarthritis in over 16s: diagnosis and management Clinical Knowledge Summaries Ankylosing spondylitis guidelines Arthritis Reasearch Council **Ankylosing Spondylitis Patient Info** Suggest link Report broken link

#### Media



#### Ankylosing spondylitis

Zero to Finals - YouTube











#### Ankylosing spondylitis

Osmosis - YouTube









#### Ankylosing spondylitis

Townsend Teaching - YouTube 6 1 0





#### Score: 22.8%

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Question 49 of 79

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A 68-year-old gentleman presents acutely with a hot, red, swollen and painful right big toe. This has happened twice before, some years ago, and he has never sought medical attention. He has a raised serum urate level and joint fluid analysis demonstrates negatively birefringent needle-shaped crystals. Once symptoms settle he is started on allopurinol but develops a severe hypersensitivity reaction to this. Which agent should be tried next as a long-term treatment option?

Colchicine	
Naproxen	
Febuxostat	
Methotrexate	
Prednisolone	

Submit answer

Reference ranges ∨

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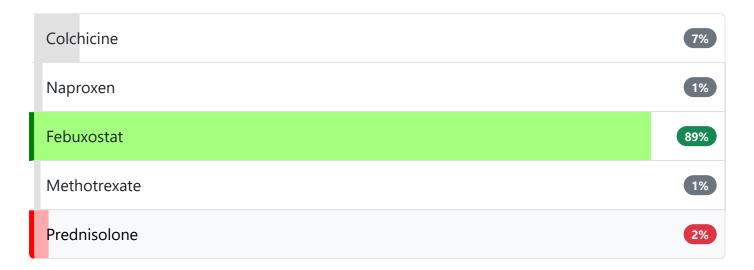
Question 49 of 79



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Febuxostat is recommended as an option for the management of chronic hyperuricaemia in gout only for patients who are intolerant of allopurinol or for whom allopurinol is contra-indicated. For the purposes of this guidance, intolerance of allopurinol is defined as adverse effects that are sufficiently severe to warrant discontinuation, or to prevent full dose escalation for optimal effectiveness.

Watch out for severe hypersensitivity reactions (including Stevens-Johnson) with febuxostat, which would be a reason to discontinue this agent.

https://www.ncbi.nlm.nih.gov/pubmed/21155617



Next question >

#### Gout: management \*

Gout is a form of microcrystal synovitis caused by the deposition of monosodium urate monohydrate in the synovium. It is caused by chronic hyperuricaemia (uric acid > 450 µmol/l)

- NSAIDs or colchicine are first-line
  - the maximum dose of NSAID should be prescribed until 1-2 days after the symptoms have settled
  - o gastroprotection (e.g. a proton pump inhibitor) may also be indicated
- colchicine
  - inhibits microtubule polymerization by binding to tubulin, interfering with mitosis. Also inhibits neutrophil motility and activity
  - has a slower onset of action
  - may be used with caution in renal impairment: the BNF advises to reduce the dose if eGFR is 10-50 ml/min and to avoid if eGFR < 10 ml/min (BNF)
  - o the main side-effect is diarrhoea
- oral steroids may be considered if NSAIDs and colchicine are contraindicated.
  - o a dose of prednisolone 15mg/day is usually used
- another option is intra-articular steroid injection
- if the patient is already taking allopurinol it should be continued

#### Indications for urate-lowering therapy (ULT)

- the British Society of Rheumatology Guidelines now advocate offering urate-lowering therapy to all patients after their **first attack of gout**
- ULT is particularly recommended if:
  - > = 2 attacks in 12 months
  - o tophi
  - o renal disease
  - uric acid renal stones
  - o prophylaxis if on cytotoxics or diuretics

#### **Urate-lowering therapy**

- it has traditionally been taught that urate-lowering therapy should not be started until 2 weeks after an acute attack, as starting too early may precipitate a further attack. The evidence base to support this however looks weak
- in 2017 the BSR updated their guidelines. They still support a delay in starting urate-lowering therapy because it is better for a patient to make long-term drug decisions whilst not in pain
  - the key passage is: 'Commencement of ULT is best delayed until inflammation has settled as ULT is better discussed when the patient is not in pain'
- allopurinol is first-line
  - $\circ~$  initial dose of 100 mg od, with the dose titrated every few weeks to aim for a serum uric acid of < 360  $\mu mol/l$
  - $\circ$  a lower target uric acid level below 300 µmol/L may be considered for patients who have tophi, chronic gouty arthritis or continue to have ongoing frequent flares despite having a uric acid below 360 µmol/L
  - o a lower initial dose of allopurinol should be given if the patient has a reduced eGFR
  - colchicine cover should be considered when starting allopurinol. NSAIDs can be used if colchicine cannot be tolerated. The BSR guidelines suggest this may need to be continued for 6 months

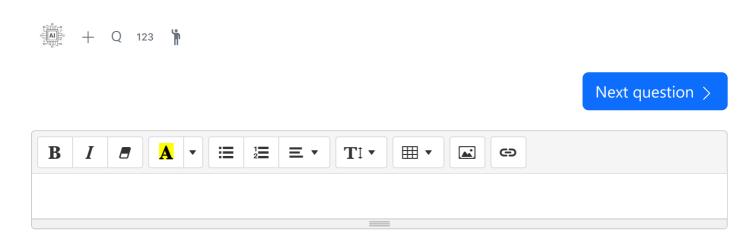
- the second-line agent when allopurinol is not tolerated or ineffective is febuxostat (also a xanthine oxidase inhibitor)
- in refractory cases other agents may be tried:
  - uricase (urate oxidase) is an enzyme that catalyzes the conversion of urate to the degradation product allantoin. It is present in certain mammals but not humans
  - in patients who have persistent symptomatic and severe gout despite the adequate use
    of urate-lowering therapy, pegloticase (polyethylene glycol modified mammalian uricase)
    can achieve rapid control of hyperuricemia. It is given as an infusion once every two
    weeks

#### Lifestyle modifications

- reduce alcohol intake and avoid during an acute attack
- lose weight if obese
- avoid food high in purines e.g. Liver, kidneys, seafood, oily fish (mackerel, sardines) and yeast products

#### Other points

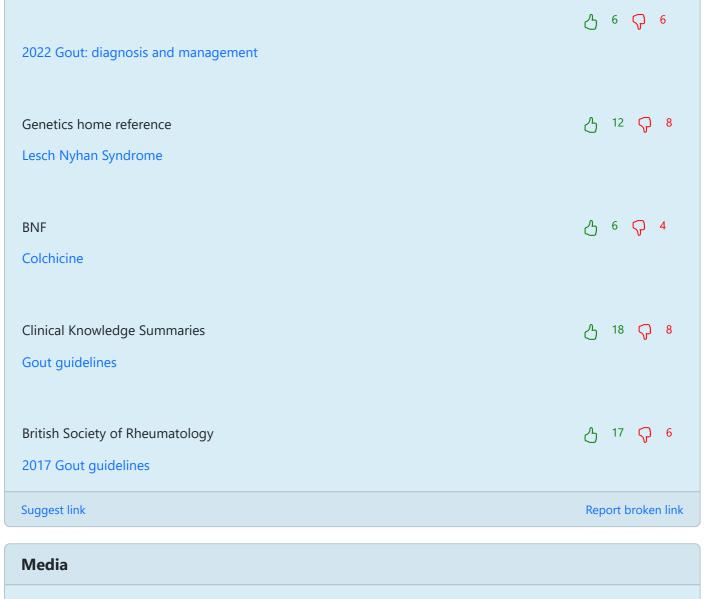
- consideration should be given to stopping precipitating drugs (such as thiazides)
- losartan has a specific uricosuric action and may be particularly suitable for the many patients who have coexistent hypertension
- increased vitamin C intake (either supplements or through normal diet) may also decrease serum uric acid levels





#### Links

**NICE** 





#### Gout

Rhesus Medicine - YouTube

→ 1 ♀ 0



#### Gout

Zero To Finals - YouTube

**分 1 分 0** 



Gout - causes, symptoms, diagnosis, treatment, pathology

Osmosis - YouTube

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78	X
79	X

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#### Question 50 of 79





A 45-year-old woman was referred to Rheumatology clinic after experiencing widespread aches and pains felt throughout her body. The pains were felt particularly in her arms and legs in addition to significant pain throughout the patient's spinal column. The patient could not recall a precise onset of her symptoms but she felt they had been present for at least 12 months, possibly longer. In addition, the patient reported on-going feelings of tiredness and lethargy. Despite going to bed around 10 pm each evening, the patient reported waking in the morning still feeling exhausted. She denied any history of hot or tender joints, skin rashes, hair loss, swallowing difficulties or dry eyes. The patient's appetite was described as normal for her with no significant change in weight.

There was no previous past medical history and the patient took no regular medications except for a non-prescription multi-vitamin. Family history was remarkable for hypothyroidism affecting her mother and elder sister. The patient worked as an accountant and lived with her two teenage children. She had separated from her ex-husband 18 months previously.

The examination did not demonstrate any evidence of active synovitis of the hands or feet with no other inflamed or deformed joints. Palpation of the muscles of the upper arms and legs as well as the paraspinal muscles was exquisitely tender. Neurological examination of the arms and legs was unremarkable. The cardiovascular and respiratory examination was unremarkable with no skin rashes.

During clinic interaction, the patient appeared tired and stressed but had a good rapport and maintained good eye contact. She denied any significant low mood but was anxious that her symptoms represented a serious underlying illness.

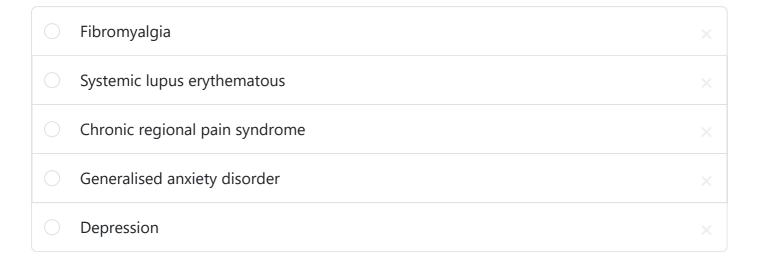
Investigations requested following clinic are listed below.

Haemoglobin	129 g / L
White cell count	7.2 * 10 <sup>9</sup> /l
Platelets	332 * 10 <sup>9</sup> /l
Mean cell volume	87 fL
Sodium	140 mmol / L
Potassium	3.6 mmol / L
Urea	3.5 mmol / L
Creatinine	68 micromol / L
Erythrocyte sedimentation rate	11 mm / h
Rheumatoid factor	Negative
Anti-nuclear antigen	Weak positive

B12	324 pmol / L (reference 74-516)		
Folate	30 nmol / L (reference 7-36)		
Serum immunoglobulin	Normal electrophoresis strip		
Thyroid stimulating hormone	0.9 microU / mL (reference 0.4-5.0)		

X-rays of hands: some minor degenerative change in right index proximal interphalangeal joint but otherwise unremarkable with no boney erosion or deformity

What is the cause of the patient's pain?



#### Submit answer

Reference ranges ∨

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Question 50 of 79



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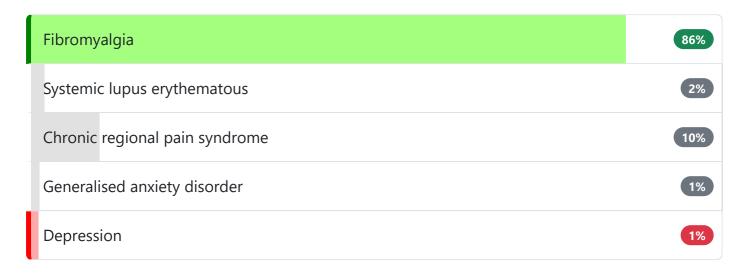
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X-rays of hands: some minor degenerative change in right index proximal interphalangeal joint but otherwise unremarkable with no boney erosion or deformity

What is the cause of the patient's pain?

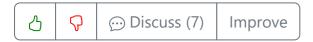


The patient has chronic widespread pain (>3 months) associated with lethargy, non-refreshing sleep and multiple tender points on palpation. Basic blood tests are essentially normal and there is no history or examination to suggest connective tissue disease or other pathology. This presentation is consistent with fibromyalgia, the diagnostic label used to describe chronic widespread pain associated with multiple muscular tender points or associated symptoms of fatigue, non-refreshing sleep or cognitive dysfunction.

Please note that many healthy individuals have weakly positive anti-nuclear antigen results and this does not imply a diagnosis of systemic lupus erythematous in the absence of symptoms and signs of the disease. It may be that requesting immunological tests was inappropriate in this patient given the lack of clinical evidence of connective tissue disease.

Chronic regional pain syndrome is associated with persistent burning pain in one limb, usually after a minor injury. The brief mental state examination documented does not suggest evidence of significant depression or generalised anxiety.

Carnes D, Underwood M, Rahman A. Fibromyalgia. BMJ 2014;348:g474.



# Fibromyalgia 🖈

Fibromyalgia is a syndrome characterised by widespread pain throughout the body with tender points at specific anatomical sites. The cause of fibromyalgia is unknown.

# Epidemiology

- women are around 5 times more likely to be affected
- typically presents between 30-50 years old

#### **Features**

- chronic pain: at multiple site, sometimes 'pain all over'
- lethargy
- cognitive impairment: 'fibro fog'
- sleep disturbance, headaches, dizziness are common

Diagnosis is clinical and sometimes refers to the American College of Rheumatology classification criteria which lists 9 pairs of tender points on the body. If a patient is tender in at least 11 of these 18 points it makes a diagnosis of fibromyalgia more likely

The management of fibromyalgia is often difficult and needs to be tailored to the individual patient. A psychosocial and multidisciplinary approach is helpful. Unfortunately there is currently a paucity of evidence and guidelines to guide practice. The following is partly based on consensus guidelines from the European League against Rheumatism (EULAR) published in 2007 and also a BMJ review in 2014.

- explanation
- aerobic exercise: has the strongest evidence base
- cognitive behavioural therapy
- medication: pregabalin, duloxetine, amitriptyline



Next question >



#### **Textbooks**

High-yield textbook

Extended textbook

# Media



# Fibromyalgia

Townsend Teaching - YouTube ♦ 2 ♀ 1





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Question 51 of 79





A 72-year-old woman attends the clinic with a 6-month history of weakness and muscle pain. On examination, you note a proximal muscle weakness of the upper (MRC grade 4/5) and lower (MRC grade 3/5) limbs. A systematic enquiry and examination were otherwise unremarkable.

Blood results are as follows:

Hb	110 g/L	Male: (135-180) Female: (115 - 160)
Platelets	310 * 10 <sup>9</sup> /L	(150 - 400)
WBC	8.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	135 mmol/L	(135 - 145)
K <sup>+</sup>	4.2 mmol/L	(3.5 - 5.0)
Urea	6.1 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)

What antibody is most sensitive (rather than specific) to the underlying condition?

Anti dsDNA antibody	
Anti-Jo-1 antibody	
Anti-PL-7 antibody	
Anti-Smith antibody	
Antinuclear antibody	

Submit answer

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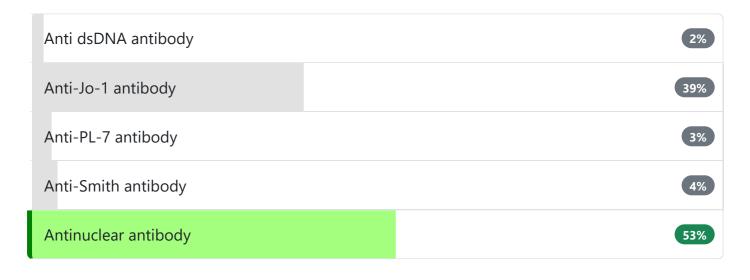


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Urea	6.1 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	2 mg/L	(< 5)

What antibody is most sensitive (rather than specific) to the underlying condition?



Proximal muscle weakness + raised CK + no rash → ?polymyositis

Important for me Less important



The patient most likely has a diagnosis of polymyositis as indicated by proximal muscle weakness and myalgia.

Antinuclear antibody (ANA) is correct. Although ANA is a relatively sensitive test, with the

majority of patients (around 80%) being positive, it is not specific to polymyositis.

**Anti-Jo-1 antibody** is incorrect. Although this antibody is highly specific to polymyositis, it is not very sensitive.

**Anti dsDNA** is incorrect. The anti-double stranded DNA antibodies (anti-dsDNA) are considered a specific marker for systemic lupus erythematosus (SLE). Although muscle weakness can occur in SLE, the absence of other diagnostic criteria of SLE (e.g. oral ulcers, fever, and neurological dysfunction) makes it a less likely diagnosis.

**Anti-PL-7 antibody** is incorrect. Although this can be positive in dermatomyositis, it is not sensitive nor specific to this condition.

**Anti-Smith** is incorrect. Anti-Smith antibody is another highly specific antibody for SLE.



Next question >

# Polymyositis \*

#### Overview

- inflammatory disorder causing symmetrical, proximal muscle weakness
- thought to be a T-cell mediated cytotoxic process directed against muscle fibres
- may be idiopathic or associated with connective tissue disorders
- associated with malignancy
- dermatomyositis is a variant of the disease where skin manifestations are prominent, for example a purple (heliotrope) rash on the cheeks and eyelids
- typically affects middle-aged, female:male 3:1

#### **Features**

- proximal muscle weakness +/- tenderness
- Raynaud's
- respiratory muscle weakness
- interstitial lung disease
  - o e.g. fibrosing alveolitis or organising pneumonia
  - seen in around 20% of patients and indicates a poor prognosis
- dysphagia, dysphonia

#### Investigations

elevated creatine kinase

- other muscle enzymes (lactate dehydrogenase (LDH), aldolase, AST and ALT) are also elevated in 85-95% of patients
- EMG
- muscle biopsy
- anti-synthetase antibodies
  - anti-Jo-1 antibodies are seen in pattern of disease associated with lung involvement,
     Raynaud's and fever

# Management

- high-dose corticosteroids tapered as symptoms improve
- azathioprine may be used as a steroid-sparing agent



Next question >

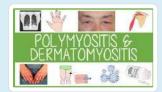


# **Textbooks**

High-yield textbook

Extended textbook

## Media



Polymyositis and Dermatomyositis in 3 Minutes

Townsend Teaching - YouTube





Zero To Finals - YouTube

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A 72-year-old woman attends rheumatology clinic for review of her osteoporosis treatment. She had been diagnosed with osteoporosis five years previously on the basis of a DEXA scan (see results below). At that time, the DEXA scan had been arranged by her GP due to a strong family history of osteoporosis (maternal hip fracture) and the patient having received multiple courses of corticosteroids as treatment for asthma. The patient has never sustained a fracture of her hip, wrist or vertebrae. Following the initial diagnosis, the patient had been treated with alendronic acid 70 mg weekly. She had not experienced any adverse effects from this medication although reported finding the need to drink copious water with her dose onerous.

The patient's past medical history was significant for asthma, although the patient reported that this was now much better controlled than previously and she had not required any corticosteroid treatment in several years. She denied any history of thyroid disease or rheumatoid arthritis. The patient had never smoked and very rarely consumed any alcohol.

Details from the patient examination in clinic and selected results from her DEXA scans are given below.

Height	160 cm
Weight	65 kg
Femoral neck BMD (5 years previously)	T -2.6 g / cm2
Femoral neck BMD (present day)	T -1.9 g / cm2
FRAX 10-year probability of major osteoporotic fracture	18 %
FRAX 10-year probability of hip fracture	6.8 %

What is the most appropriate management of the patient's osteoporosis?

Discontinue alendronic acid and initiate treatment with denosumab	
Hold further osteoporosis treatment with repeat DEXA scan in two years	
Continue treatment with alendronic acid with repeat DEXA scan in five years	
Continue treatment with alendronic acid with repeat DEXA scan in two years	
Hold further osteoporosis treatment with repeat DEXA scan in five years	

Submit answer

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Question 52 of 79

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Femoral neck BMD (present day)	T -1.9 g / cm2
FRAX 10-year probability of major osteoporotic fracture	18 %
FRAX 10-year probability of hip fracture	6.8 %

What is the most appropriate management of the patient's osteoporosis?

Discontinue alendronic acid and initiate treatment with denosumab	12%
Hold further osteoporosis treatment with repeat DEXA scan in two years	29%
Continue treatment with alendronic acid with repeat DEXA scan in five years	27%
Continue treatment with alendronic acid with repeat DEXA scan in two years	26%
Hold further osteoporosis treatment with repeat DEXA scan in five years	6%

At the present time it is uncertain how long patients should take bisphosphonates. Recent concerns about side effects of long-term bisphosphonate treatment (for example, atypical femur

fractures) have led to recommendations about the use of treatment breaks ('drug holidays') in certain patients.

The patient in this example meets the recommended criteria for a treatment break. In particular, her age (< 75 years), femoral neck bone mineral density > -2.5 and lack of history of fragility fracture are all favourable. A repeat DEXA scan is recommended after two years or in the event of fragility fracture to review need for further treatment.

It is recommended to use the WHO Fracture Risk Assessment Tool (FRAX) to estimate a patient's 10-year risk of fragility fracture. This can be combined National Osteoporosis Guideline Group (NOGG) guidance to inform treatment decisions. In this example, the patient's FRAX score would place her below the treatment threshold of NOGG guidance. It should be noted, that despite the recommendation to use FRAX score and NOGG guidance in this situation, neither tools are validated for patients taking bisphosphonates.

Parenteral treatments for osteoporosis such as denosumab should be reserved for individuals in whom bisphosphonate treatment has failure, or who are intolerant of bisphosphonates.

Paskins Z, Warburton L. Bisphosphonates beyond five years. BMJ 2016;352:i264.

https://www.shef.ac.uk/FRAX/tool.jsp



Next question >

# Osteoporosis: management \*

The National Osteoporosis Guideline Group (NOGG) updated their guidelines in 2021. NICE guidelines also have a section on the management of osteoporosis, largely based on the NOGG guidelines. Remember that osteoporosis is usually asymptomatic until a fracture occurs. When thinking about osteoporosis management it is useful to think about a number of potential clinical scenarios:

- a patient who has been identified as being at high risk of a fragility fracture based on a QFracture or FRAX score (please see the textbook entry on 'Osteoporosis: assessing risk')
- a patient who is about to start treatment that puts them at significant risk of developing osteoporosis the most common example is longer-term glucocorticoids
- a patient who has just had a fragility fracture e.g. a symptomatic osteoporotic vertebral fracture

# **General management points**

General points about the management of all patients

- all patients who are at risk of osteoporosis or have osteoporosis should be given advice regarding:
  - lifestyle changes: a healthy, balanced diet, moderation of alcohol consumption and avoidance of smoking
  - o a sufficient dietary calcium and vitamin D intake: supplementation should be offered to all women unless the clinician is confident they have adequate calcium intake and are vitamin D replete
  - o encourage a combination of regular weight-bearing and muscle strengthening exercise
- secondary causes of osteoporosis should be considered and treated
  - e.g. hypogonadism in women or men e.g. hormone replacement therapy for premature menopause
- bisphosphonates are the first-line drug treatment for patients at risk of fragility fractures
  - oral bisphosphonates such as alendronate and risedronate are typically first-line. These are often taken weekly are need taking in a particular way to minimise the risk of oesophageal side-effects
  - however, the NOGG recommend IV zoledronate as the first-line treatment following a hip fracture. This is given yearly
- denosumab is generally used as a second-line treatment
- other possible treatment options include:
  - o strontium ranelate
  - raloxifene
  - o teriparatide
  - romosozumab

# Clinical scenarios

Fragility risk fracture assessment

- if a patient is deemed high-risk based on a QFracture or FRAX score they should have a DEXA scan to assess bone mineral density (BMD)
  - the BMD threshold for defining osteoporosis is a T-score of 2.5 SD or below
  - o some patients may not be suitable for BMD assessment due to frailty etc.
- general osteoporosis management as above
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

Postmenopausal women, and men age ≥50, who are treated with oral glucocorticoids:

- if starting ≥7.5 mg/day prednisolone or equivalent for the next 3 months, start bone protective treatment at the same time
- general osteoporosis management as above
- don't wait for a DEXA scan before starting treatment
- oral bisphosphonates are used first-line e.g. alendronate or risedronate

A postmenopausal woman, or a man age  $\geq 50$  has a symptomatic osteoporotic vertebral fracture:

• general osteoporosis management as above

 start treatment straight away - oral bisphosphonates are used first-line e.g. alendronate or risedronate

#### Hip fracture in older adults

- in older adults a hip fracture is a manifestation of osteoporosis
  - following a fragility fracture in women ≥ 75 years, a DEXA scan is not necessary to diagnose osteoporosis and hence commence a bisphosphonate
  - BMD should be measured, but this acts as a baseline rather than determining whether treatment should be given
- bisphosphonates should be given first-line
  - NOGG recommends IV zoledronate but local guidelines may vary and oral bisphosphonates are often used

# Follow-up

Plan to prescribe oral bisphosphonates for at least 5 years, or intravenous bisphosphonates for at least 3 years and then re-assess fracture risk.

# Supplementary notes on treatment

# Bisphosphonates

- bisphosphonates bind to hydroxyapatite in bone, inhibiting osteoclast-mediated bone resorption
- common side effects include gastrointestinal discomfort, oesophagitis, and hypocalcaemia. Atypical femoral fractures and osteonecrosis of the jaw are rare but serious risks.
- available in oral and intravenous formulations. Oral bisphosphonates should be taken with a full glass of water, on an empty stomach, and the patient should remain upright for at least 30 minutes afterwards.

#### Denosumab

- human monoclonal antibody that inhibits RANK ligand, which in turn inhibits the maturation of osteoclasts
- also used for cancer patients with bone metastases to reduce skeletal-related events.
- given as a single subcutaneous injection every 6 months

#### Raloxifene

- selective oestrogen receptor modulator (SERM)
- has been shown to prevent bone loss and to reduce the risk of vertebral fractures, but has not yet been shown to reduce the risk of non-vertebral fractures
- has been shown to increase bone density in the spine and proximal femur
- may worsen menopausal symptoms
- increased risk of thromboembolic events
- may decrease the risk of breast cancer

#### Strontium ranelate

- 'dual action bone agent' increases deposition of new bone by osteoblasts (promotes differentiation of pre-osteoblast to osteoblast) and reduces the resorption of bone by inhibiting osteoclasts
- concerns regarding the safety profile of strontium have been raised recently. It should only be prescribed by a specialist in secondary care
- due to these concerns the European Medicines Agency in 2014 said it should only be used by people for whom there are no other treatments for osteoporosis
- increased risk of cardiovascular events: any history of cardiovascular disease or significant risk of cardiovascular disease is a contraindication
- increased risk of thromboembolic events: a Drug Safety Update in 2012 recommended it is not used in patients with a history of venous thromboembolism
- may cause serious skin reactions such as Stevens Johnson syndrome

## Teriparatide

- recombinant form of parathyroid hormone
- very effective at increasing bone mineral density but role in the management of osteoporosis yet to be clearly defined

#### Romosozumab

- a monoclonal antibody that inhibits sclerostin, thereby increasing bone formation and decreasing bone resorption
- this dual action significantly improves bone density and reduces fracture risk.



© Image used on license from Radiopaedia

MRI showing osteoporotic fractures of the 10th and 12th thoracic vertebrae.



Next question >

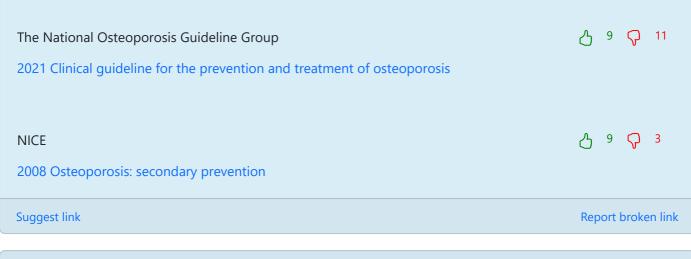


# **Textbooks**

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# Links



# Media



Osteoporosis pharmacology, prevention and treatment

Armando Hasudungan - YouTube







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# Question 53 of 79





A 58-year-old man who has no fixed abode comes to the Emergency department because he is unable to walk. He has a history of alcoholism and type 2 diabetes. His main complaint is that his shoes have worn out and because of loss of sensation he didn't notice that he had stepped on a nail. In total the lesion on his right foot has been present for approximately 3 weeks.

Which of the following is the next step in evaluating his foot injury?

Inflammatory markers	
MRI foot	
Plain x-ray foot	
USS foot	
Wound swab	

# Submit answer

Reference ranges ∨

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Question 53 of 79

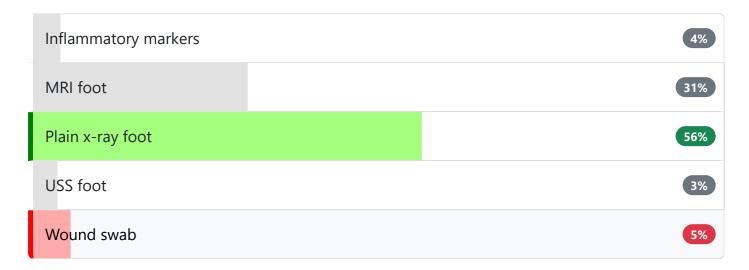


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Which of the following is the next step in evaluating his foot injury?



The key next step here is to gather useful information about the extent of any foot infection. By 3 weeks post injury, changes consistent with osteomyelitis should be visible on plain x-ray. These may include soft tissue swelling, bone demineralisation, cortical irregularity, and an elevated periosteum.

Many patients progress from a plain x-ray on to MRI imaging of the foot for further evaluation of the extent of infection and to guide potential operative approaches for debridement. Inflammatory markers are a very non-specific marker of infection and ultrasound of the foot is only useful to visualise soft tissue swelling or collection of pus / fluid. A wound swab is likely to show a range of bacteria, this is what drives selection of a broader spectrum antibiotic such as co-amoxiclav in the diabetic population.



Next question >

# Osteomyelitis \*

Osteomyelitis describes an infection of the bone. It may be subclassified into:

- haematogenous osteomyelitis
  - o results from bacteraemia

- o is usually monomicrobial
- o most common form in children
- vertebral osteomyelitis is the most common form of haematogenous osteomyelitis in adults
- risk factors include: sickle cell anaemia, intravenous drug user, immunosuppression due to either medication or HIV, infective endocarditis
- non-haematogenous osteomyelitis:
  - results from the contiguous spread of infection from adjacent soft tissues to the bone or from direct injury/trauma to bone
  - o is often polymicrobial
  - o most common form in adults
  - risk factors include: diabetic foot ulcers/pressure sores, diabetes mellitus, peripheral arterial disease

# Microbiology

• *Staph. aureus* is the most common cause except in patients with sickle-cell anaemia where *Salmonella* species predominate

# Investigations

• MRI is the imaging modality of choice, with a sensitivity of 90-100%

# Management

- flucloxacillin for 6 weeks
- clindamycin if penicillin-allergic



Next question >



# High-yield textbook

Extended textbook

**Textbooks** 



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#### Question 54 of 79



 $\Rightarrow$ 

A 32-year-old woman with a history of systemic lupus erythematosus and stable joint disease currently managed with hydroxychloroquine comes to the nephrology clinic. She complains of worsening peripheral oedema. Examination reveals a blood pressure of 159/88 mmHg, her pulse is 78 beats per minute and regular. There is pitting oedema of both lower limbs.

# investigations

Na <sup>+</sup>	141 mmol/l
K <sup>+</sup>	5.1 mmol/l
Urea	8.1 mmol/l
Creatinine	159 µmol/l
Creatinine one month earlier	132 µmol/l
Urinary protein	5 g/24hrs

Which of the following is the most appropriate intervention with respect to long-term renal outcomes?

Cyclophosphamide	
Methotrexate	
Methylprednisolone	
Mycophenolate	
Rituximab	

Submit answer

Reference ranges ∨

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Question 54 of 79



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## investigations

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K <sup>+</sup>	5.1 mmol/l
Urea	8.1 mmol/l
Creatinine	159 µmol/l
Creatinine one month earlier	132 µmol/l
Urinary protein	5 g/24hrs

Which of the following is the most appropriate intervention with respect to long-term renal outcomes?

Cyclophosphamide	28%
Methotrexate	6%
Methylprednisolone	13%
Mycophenolate	50%
Rituximab	3%

Mycophenolate mofetil (MMF) is a prodrug of mycophenolic acid (MPA), an inhibitor of inosine-5'-monophosphate dehydrogenase. MPA depletes guanosine nucleotides preferentially in T and B lymphocytes and inhibits their proliferation. In proteinuric renal disease related to systemic lupus erythematosus, it is the intervention of choice.

Cyclophosphamide is not recommended for lupus nephritis because renal outcomes are similar to those for mycophenolate, with higher rates of infection. Although corticosteroids are used in conjunction with second-line agents, it is mycophenolate which is more important with respect to long-term renal outcomes. Anti-B cell agents such as rituximab are used in the treatment of lupus but are normally added to therapy with a conventional second line agent such as mycophenolate. Methotrexate is used primarily in the treatment of rheumatoid arthritis.

Next question >

# Systemic lupus erythematosus: features \*

Systemic lupus erythematosus (SLE) is a multisystem, autoimmune disorder. It typically presents in early adulthood and is more common in women and people of Afro-Caribbean origin.

## General features

- fatique
- fever
- mouth ulcers
- lymphadenopathy

#### Skin

- malar (butterfly) rash: spares nasolabial folds
- discoid rash: scaly, erythematous, well demarcated rash in sun-exposed areas. Lesions may progress to become pigmented and hyperkeratotic before becoming atrophic
- photosensitivity
- Raynaud's phenomenon
- livedo reticularis
- non-scarring alopecia

#### Musculoskeletal

- arthralgia
- non-erosive arthritis

#### Cardiovascular

- pericarditis: the most common cardiac manifestation
- myocarditis

## Respiratory

- pleurisy
- fibrosing alveolitis

#### Renal

- proteinuria
- glomerulonephritis (diffuse proliferative glomerulonephritis is the most common type)

# Neuropsychiatric

- anxiety and depression
- psychosis
- seizures

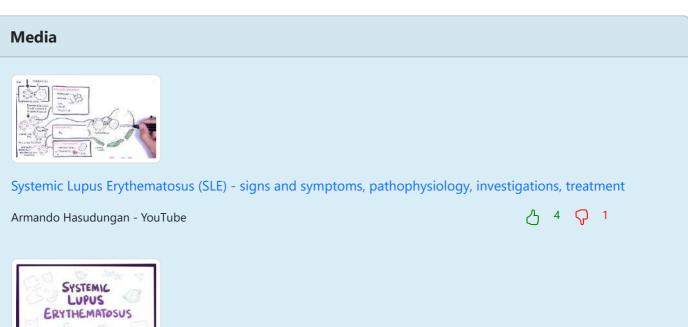


Next question >









Osmosis - YouTube

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Question 55 of 79





A 62-year-old man with a diagnosis of Paget's disease is seen in clinic with a two month history of worsening bone pain, mainly in his left leg. His medications include paracetamol, ibuprofen, and alendronate.

Examination reveals marked deformity of the long bones, particularly the left tibia.

#### Blood tests:

Calcium	2.40 mmol/L (2.25-2.5)
Albumin	37g/L (34-54)
Corrected calcium	2.50 mmol/L (2.25-2.5)
Alkaline phosphatase	484 U/L (45-105)
Alanine transaminase	27 U/L (5-35)

What is the next stage in the treatment of this patient?

Cholecalciferol	
Surgery	
Calcitonin	
Hearing aid	
Prednisolone	

Submit answer

Reference ranges \

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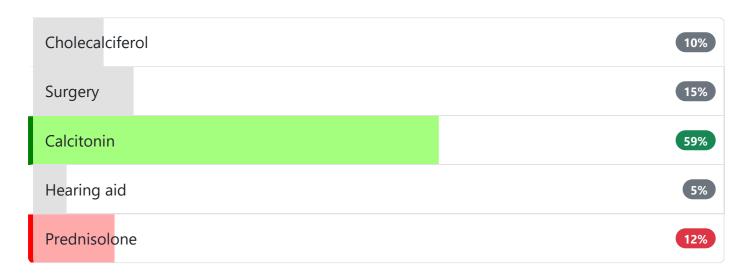
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Alkaline phosphatase	484 U/L (45-105)
Alanine transaminase	27 U/L (5-35)

What is the next stage in the treatment of this patient?



Paget's disease is characterised by abnormal bone remodelling, particularly in the skull and long bones. The characteristic blood test results include an elevated alkaline phosphatase, with otherwise normal liver function tests (as alkaline phosphatase is also found in bone). A raised calcium may only be seen if there is associated immobility.

Analgesics and non-steroidal inflammatory drugs are initially used to manage pain, with treatment escalated to bisphosphonates and calcitonin in refractory cases.

#### Reference

Selby et al. Guidelines on the management of Paget's disease of bone. Bone, 2002; 31:36673.

Next question >

# Paget's disease of the bone \*

Paget's disease is a disease of increased but uncontrolled bone turnover. It is thought to be primarily a disorder of osteoclasts, with excessive osteoclastic resorption followed by increased osteoblastic activity. Paget's disease is common (UK prevalence 5%) but symptomatic in only 1 in 20 patients. The skull, spine/pelvis, and long bones of the lower extremities are most commonly affected.

## Predisposing factors

- increasing age
- male sex
- northern latitude
- family history

Clinical features - only 5% of patients are symptomatic

- the stereotypical presentation is an older male with bone pain and an isolated raised ALP
- bone pain (e.g. pelvis, lumbar spine, femur)
- classical, untreated features: bowing of tibia, bossing of skull

#### Investigations

- bloods
  - raised alkaline phosphatase (ALP)
  - calcium and phosphate are typically normal. Hypercalcaemia may occasionally occur with prolonged immobilisation
- other markers of bone turnover include
  - procollagen type I N-terminal propeptide (PINP)
  - serum C-telopeptide (CTx)
  - urinary N-telopeptide (NTx)
  - urinary hydroxyproline
- x-rays
  - osteolysis in early disease → mixed lytic/sclerotic lesions later
  - o skull x-ray: thickened vault, osteoporosis circumscripta
- bone scintigraphy
  - increased uptake is seen focally at the sites of active bone lesions

#### Management

- indications for treatment include
  - bone pain

- o skull or long bone deformity
- o fracture
- o periarticular Paget's
- bisphosphonate (either oral risedronate or IV zoledronate)
- calcitonin is less commonly used now

# Complications

- deafness (cranial nerve entrapment)
- bone sarcoma (1% if affected for > 10 years)
- fractures
- skull thickening
- high-output cardiac failure









Next question >









2019 Paget's disease of the bone guidelines

Suggest link Report broken link

# Media



# Paget's Disease of the bone

Armando Hasudungan - YouTube 6 0 0 0









# Paget's disease of the bone

Osmosis - YouTube







Report broken media

Score: 22.8%

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Question 56 of 79

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 $\overline{\Rightarrow}$ 

A 35-year-old lady with diffuse systemic sclerosis attends the rheumatology clinic. She has had worsening arthralgia over the last 2 months, mainly in the hands and feet. She does not complain of any other symptoms.

On examination her blood pressure is 161/94 mmHg, her heart rate is 90 beats per minute and her oxygen saturations are 96% on room air. She has sclerodactyly and tender small joints of the hands and feet with mild swelling. The hands are pale and cool. Her chest is clear.

Her blood tests are as follows:

Hb	110 g/l	Na <sup>+</sup>	136 mmol/l	Bilirubin	5 µmol/l
Platelets	210 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.7 mmol/l	ALP	90 u/l
WBC	10 * 10 <sup>9</sup> /l	Urea	5 mmol/l	ALT	21 u/l
Neuts	7 * 10 <sup>9</sup> /l	Creatinine	89 µmol/l	γGT	30 u/l
Lymphs	2.5 * 10 <sup>9</sup> /l	ESR	99 mm/h	Albumin	32 g/l

Which drug should be used with caution in this patient?

Tacrolimus	
Azathioprine	
Methotrexate	
Mycophenolate mofetil	
Prednisolone	

Submit answer

Reference ranges ∨

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Question 56 of 79



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Lymphs	2.5 * 10 <sup>9</sup> /l	ESR	99 mm/h	Albumin	32 g/l

Which drug should be used with caution in this patient?



Steroid use is known to precipitate scleroderma renal crisis and this is a patient who already has hypertension. Azathioprine, mycophenolate mofetil, tacrolimus and methotrexate are all immunosuppressive agents which may be used in rheumatological conditions, though methotrexate may cause additional pulmonary fibrosis.

Reference: Denton CP. Renal manifestations of systemic sclerosis - clinical features and outcome assessment. Rheumatology 2008;47:v54-v56.



Systemic sclerosis is a condition of unknown aetiology characterised by hardened, sclerotic skin and other connective tissues. It is four times more common in females.

There are three patterns of disease:

Limited cutaneous systemic sclerosis

- Raynaud's may be the first sign
- scleroderma affects face and distal limbs predominately
- associated with anti-centromere antibodies
- a subtype of limited systemic sclerosis is CREST syndrome: Calcinosis, Raynaud's phenomenon, oEsophageal dysmotility, Sclerodactyly, Telangiectasia

Diffuse cutaneous systemic sclerosis

- scleroderma affects trunk and proximal limbs predominately
- associated with anti scl-70 antibodies
- the most common cause of death is now respiratory involvement, which is seen in around 80%: interstitial lung disease (ILD) and pulmonary arterial hypertension (PAH)
- other complications include renal disease and hypertension
  - o patients with renal disease should be started on an ACE inhibitor
- poor prognosis

Scleroderma (without internal organ involvement)

- tightening and fibrosis of skin
- may be manifest as plaques (morphoea) or linear







### **Antibodies**

- ANA positive in 90%
- RF positive in 30%
- anti-scl-70 antibodies associated with diffuse cutaneous systemic sclerosis
  - associated with a higher risk of severe interstitial lung disease
- anti-centromere antibodies associated with limited cutaneous systemic sclerosis



Next question >





Suggest link Report broken link





## Scleroderma

Townsend Teaching - YouTube 4 Q 0



### Scleroderma

Osmosis - YouTube





# Systemic Sclerosis and Scleroderma

Zero to Finals - YouTube





Report broken media

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#### Question 57 of 79





A 65-year-old man was referred to rheumatology for advice regarding the management of his gout. The patient had suffered intermittent episodes of inflammation of the first metatarsophalangeal joint of both feet over the past ten years. The frequency of these episodes had been increasing, with 6 episodes in the past year. In addition, the patient's right knee had recently become inflamed with microscopy of synovial aspirate demonstrating needle shaped crystals with negative birefringence.

Colchicine and NSAIDs had been used effectively to provide symptomatic relief to the patient during an acute attack. Allopurinol had been previously trialled as prophylaxis at a dose of 200 mg daily, although was stopped after the patient's renal function was noted to have deteriorated after allopurinol was initiated. Lifestyle modifications have also been attempted.

Other medical problems included type 2 diabetes, hypertension, hypercholesterolaemia and chronic renal failure. Regular medications were ramipril, metformin and simvastatin.

On examination the patient was noted to be obese without evidence of current joint inflammation. Tophi were noted on examination of the patient's ears. Blood tests taken prior to clinic attendance are listed below.

Hb	16.5 g/dl
Platelets	150 * 10 <sup>9</sup> /l
WBC	8.6 * 10 <sup>9</sup> /l

Na <sup>+</sup>	137 mmol/l
K <sup>+</sup>	4.7 mmol/l
Urea	11.2 mmol/l
Creatinine	190 µmol/l
eGFR	45 ml/min
Calcium (adjusted)	2.3 mmol/l
Urate	395 µmol/l

What is the best strategy for gout prophylaxis in this patient?

Prednisolone 10 mg daily	
Colchicine	

Febuxostat	
Naproxen	
Reduced dose allopurinol	

# Submit answer

Reference ranges  $\checkmark$ 

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Question 57 of 79



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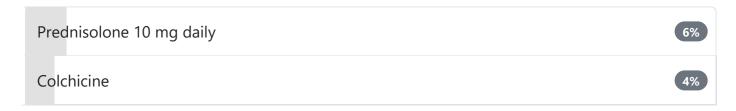
Other medical problems included type 2 diabetes, hypertension, hypercholesterolaemia and chronic renal failure. Regular medications were ramipril, metformin and simvastatin.

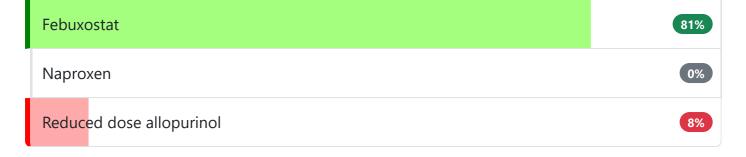
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Platelets	150 * 10 <sup>9</sup> /l
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Urate	395 μmol/l

What is the best strategy for gout prophylaxis in this patient?



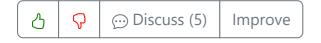


Urate lowering therapy is indicated in recurrent attacks of acute gout, although there are no firm guidelines as to when to initiate therapy. Target serum uric acid levels are usually taken as less than 360 micromol / L. Febuxostat is a non-purine xanthine oxidase inhibitor approved by NICE for use in individuals, such as this patient, who are intolerant of allopurinol or in whom allopurinol is contra-indicated.

Prednisolone, colchicine and naproxen are all used in the acute treatment of gout. Extended use of colchicine and NSAIDs can be considered to reduce the risk of gout relapse during the instigation of allopurinol therapy but have no role as prophylactic therapy in isolation.

Allopurinol can cause renal, hepatic and severe skin reactions (allopurinol hypersensitivity syndrome) and is best avoided in this patient given previous worsening of renal function with a relatively low dose of allopurinol.

Roddy E, Mallen C, Doherty M. Gout. BMJ 2013;347:5648.



Next question >

# Gout: management \*

Gout is a form of microcrystal synovitis caused by the deposition of monosodium urate monohydrate in the synovium. It is caused by chronic hyperuricaemia (uric acid >  $450 \mu mol/l$ )

### Acute management

- NSAIDs or colchicine are first-line
  - the maximum dose of NSAID should be prescribed until 1-2 days after the symptoms have settled
  - o gastroprotection (e.g. a proton pump inhibitor) may also be indicated
- colchicine
  - inhibits microtubule polymerization by binding to tubulin, interfering with mitosis. Also inhibits neutrophil motility and activity
  - has a slower onset of action
  - may be used with caution in renal impairment: the BNF advises to reduce the dose if eGFR is 10-50 ml/min and to avoid if eGFR < 10 ml/min (BNF)

- o the main side-effect is diarrhoea
- oral steroids may be considered if NSAIDs and colchicine are contraindicated.
  - o a dose of prednisolone 15mg/day is usually used
- another option is intra-articular steroid injection
- if the patient is already taking allopurinol it should be continued

## Indications for urate-lowering therapy (ULT)

- the British Society of Rheumatology Guidelines now advocate offering urate-lowering therapy to all patients after their **first attack of gout**
- ULT is particularly recommended if:
  - > = 2 attacks in 12 months
  - o tophi
  - o renal disease
  - uric acid renal stones
  - o prophylaxis if on cytotoxics or diuretics

# **Urate-lowering therapy**

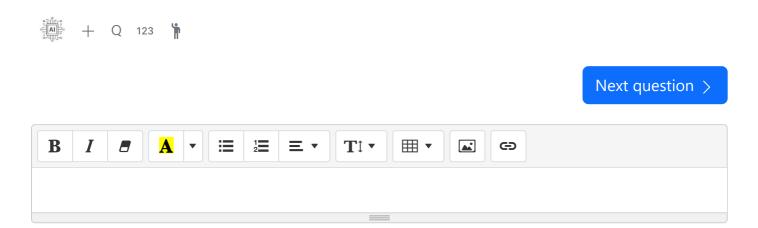
- it has traditionally been taught that urate-lowering therapy should not be started until 2 weeks after an acute attack, as starting too early may precipitate a further attack. The evidence base to support this however looks weak
- in 2017 the BSR updated their guidelines. They still support a delay in starting urate-lowering therapy because it is better for a patient to make long-term drug decisions whilst not in pain
  - the key passage is: 'Commencement of ULT is best delayed until inflammation has settled as ULT is better discussed when the patient is not in pain'
- allopurinol is first-line
  - $\circ$  initial dose of 100 mg od, with the dose titrated every few weeks to aim for a serum uric acid of < 360  $\mu$ mol/l
  - $\circ$  a lower target uric acid level below 300 µmol/L may be considered for patients who have tophi, chronic gouty arthritis or continue to have ongoing frequent flares despite having a uric acid below 360 µmol/L
  - o a lower initial dose of allopurinol should be given if the patient has a reduced eGFR
  - colchicine cover should be considered when starting allopurinol. NSAIDs can be used if colchicine cannot be tolerated. The BSR guidelines suggest this may need to be continued for 6 months
- the second-line agent when allopurinol is not tolerated or ineffective is febuxostat (also a xanthine oxidase inhibitor)
- in refractory cases other agents may be tried:
  - uricase (urate oxidase) is an enzyme that catalyzes the conversion of urate to the degradation product allantoin. It is present in certain mammals but not humans
  - in patients who have persistent symptomatic and severe gout despite the adequate use of urate-lowering therapy, pegloticase (polyethylene glycol modified mammalian uricase) can achieve rapid control of hyperuricemia. It is given as an infusion once every two weeks

## Lifestyle modifications

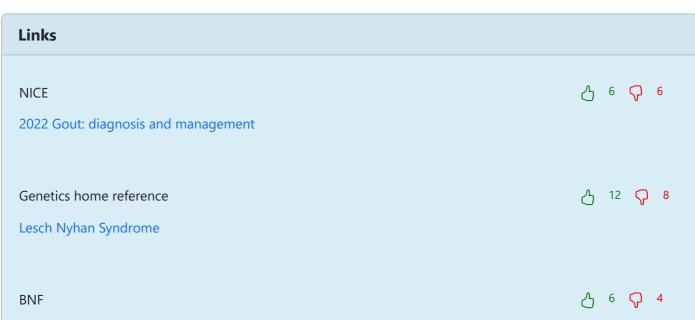
- reduce alcohol intake and avoid during an acute attack
- lose weight if obese
- avoid food high in purines e.g. Liver, kidneys, seafood, oily fish (mackerel, sardines) and yeast products

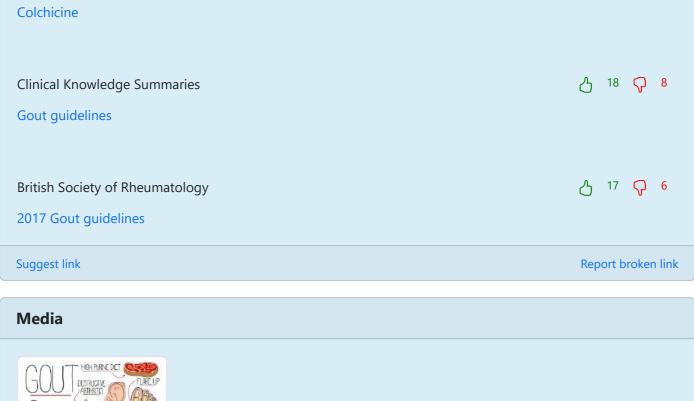
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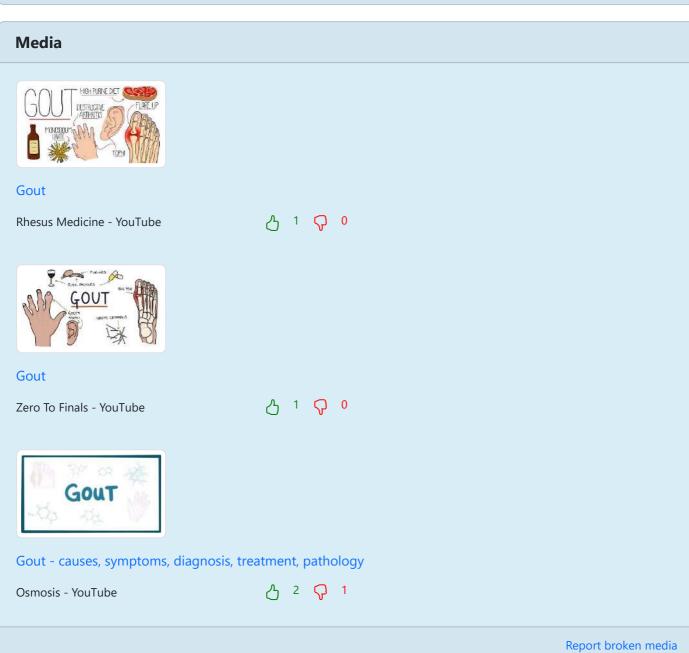
- consideration should be given to stopping precipitating drugs (such as thiazides)
- losartan has a specific uricosuric action and may be particularly suitable for the many patients who have coexistent hypertension
- increased vitamin C intake (either supplements or through normal diet) may also decrease serum uric acid levels











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# Question 58 of 79





A 45-year-old female presents with severe lower back pain. She states that the pain started a couple of months ago and has progressively got worse. She has also been experiencing fevers, rigors and weight loss. On examination she has severe tenderness on palpation over the L4 vertebrae. There is no associated neurological signs.

Blood results are as follows:

Hb	105 g/l	Na <sup>+</sup>	140 mmol/l
Platelets	542 * 10 <sup>9</sup> /I	K <sup>+</sup>	3.8 mmol/l
WBC	20.2 * 10 <sup>9</sup> /l	Urea	8.5 mmol/l
Neuts	15.4 * 10 <sup>9</sup> /l	Creatinine	92 µmol/l
Lymphs	2.2 * 10 <sup>9</sup> /l	CRP	288 mg/l

What investigation is most likely to confirm the diagnosis?

Plane film lumbar x-ray	
MRI	
CT with bone window	
White cell scan	
Blood culture	

Submit answer

Reference ranges ∨

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Question 58 of 79



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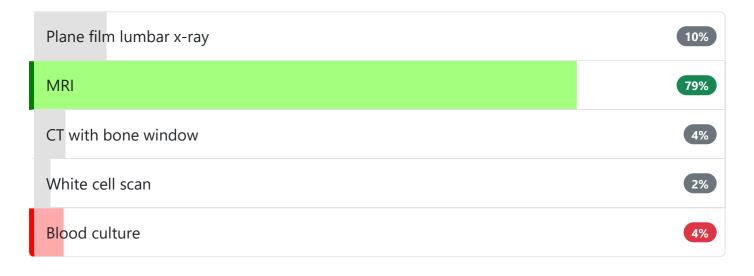
 $\Rightarrow$ 

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Neuts	15.4 * 10 <sup>9</sup> /l	Creatinine	92 µmol/l
Lymphs	2.2 * 10 <sup>9</sup> /l	CRP	288 mg/l

What investigation is most likely to confirm the diagnosis?



An MRI scan of the spine is the most appropriate investigation to diagnose infected discitis as it has the greatest sensitivity

Important for me Less important



The patient has systemic signs indicating infection, together with point tenderness over a vertebrae, making discitis the most likely diagnosis. Discitis, or vertebral osteomyelitis, is defined as infection of vertebral bones with extension into intervertebral discs. The clinical presentation of discitis can be variable from acute back pain and fevers to slowly progressive back pain over a number of months.

More than 50% of discitis cases are due to Staphylococcus aureus. Infection may also be due to a

wide range of other organisms including gram negatives, *Streptococci* and Tuberculosis. Mixed infection is also possible. Three sets of blood cultures should be taken before anti-microbials are given. However, blood cultures are only positive in approximately 50-70% of patients with discitis.

MRI spine is the most sensitive diagnostic test. A whole spine MRI should be performed as discitis frequently occurs at multiple levels. MRI is the imaging modality of choice due to its very high sensitivity and specificity. It is also useful in differentiating between pyogenic, tuberculous, and fungal infections, and a neoplastic process.

A bone scan and white cell (WBC) scan may be used to demonstrate increased uptake at the site of infection, and are more sensitive than plain film and CT, but lack specificity.

CT guided biopsy should be performed in all patients prior to initiation of antibiotics unless there is a positive

blood culture which fits the clinical picture or the patient is septic/unstable.



Next question >

# Discitis \*

Discitis is an infection in the intervertebral disc space. It can lead to serious complications such as sepsis or an epidural abscess.

#### **Features**

- Back pain
- General features
  - o pyrexia,
  - o rigors
  - sepsis
- Neurological features
  - e.g. changing lower limb neurology
  - if an epidural abscess develops

## Causes

- Bacterial
  - Staphylococcus aureus is the most common cause of discitis
- Viral
- TB
- Aseptic

# Diagnosis

- Imaging: MRI has the highest sensitivity
- CT-guided biopsy may be required to guide antimicrobial treatment



## **Treatment**

- The standard therapy requires six to eight weeks of intravenous antibiotic therapy
- Choice of antibiotic is dependent on a variety of factors. The most important factor is to identify the organism with a positive culture (e.g. blood culture, or CT-guided biopsy)
- the patient should be assessed for endocarditis e.g. with transthoracic echo or transesophageal echo. Discitis is usually due to haematogenous seeding of the vertebrae implying that the patient has had a bacteraemia and seeding could have occurred elsewhere

# Complications

- sepsis
- epidural abscess



Next question >





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Question 59 of 79





A 76-year-old man presents to his GP with a 3-month history of generalised weakness and fatigue. He is otherwise well with a past medical history of osteoarthritis, gout and type 2 diabetes mellitus (T2DM). His medications include paracetamol and metformin. He is an ex-smoker of 20 cigarettes per day.

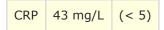
On examination, he has relative weakness of shoulder abduction and adduction and needs to use his arms to get up out of the chair, though distal power is maintained. His eye movements are normal and reflexes are preserved throughout, sensation is also normal. There is no obvious rash or joint swelling.

#### Blood tests show:

Hb	121 g/L	Male: (135-180) Female: (115 - 160)
Platelets	231 * 10 <sup>9</sup> /L	(150 - 400)
WBC	9.4 * 10 <sup>9</sup> /L	(4.0 - 11.0)

Na <sup>+</sup>	143 mmol/L	(135 - 145)
K <sup>+</sup>	4.8 mmol/L	(3.5 - 5.0)
Bicarbonate	28 mmol/L	(22 - 29)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	136 µmol/L	(55 - 120)

Thyroid stimulating hormone (TSH)	2.3 mU/L	(0.5-5.5)
Free thyroxine (T4)	11.7 pmol/L	(9.0 - 18)
Creatine kinase	632 U/L	(35 - 250)



# What is the likely underlying diagnosis?

Dermatomyositis	
Lambert Eaton myasthenic syndrome	
Myasthenia gravis	

Polymyalgia rheumatica	
Polymyositis	

# Submit answer

Reference ranges ✓

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Question 59 of 79



 $\Box$ 



A 76-year-old man presents to his GP with a 3-month history of generalised weakness and fatigue. He is otherwise well with a past medical history of osteoarthritis, gout and type 2 diabetes mellitus (T2DM). His medications include paracetamol and metformin. He is an ex-smoker of 20 cigarettes per day.

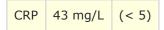
On examination, he has relative weakness of shoulder abduction and adduction and needs to use his arms to get up out of the chair, though distal power is maintained. His eye movements are normal and reflexes are preserved throughout, sensation is also normal. There is no obvious rash or joint swelling.

#### Blood tests show:

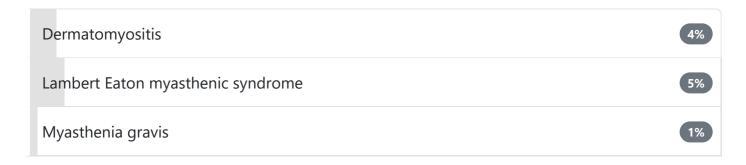
Hb	121 g/L	Male: (135-180) Female: (115 - 160)
Platelets	231 * 10 <sup>9</sup> /L	(150 - 400)
WBC	9.4 * 10 <sup>9</sup> /L	(4.0 - 11.0)

Na <sup>+</sup>	143 mmol/L	(135 - 145)
K <sup>+</sup>	4.8 mmol/L	(3.5 - 5.0)
Bicarbonate	28 mmol/L	(22 - 29)
Urea	6.5 mmol/L	(2.0 - 7.0)
Creatinine	136 µmol/L	(55 - 120)

Thyroid stimulating hormone (TSH)	2.3 mU/L	(0.5-5.5)
Free thyroxine (T4)	11.7 pmol/L	(9.0 - 18)
Creatine kinase	632 U/L	(35 - 250)



# What is the likely underlying diagnosis?



Proximal muscle weakness + raised CK + no rash → ?polymyositis

Important for me Less important



This patient has proximal muscle weakness without pain and raised creatinine kinase (CK), which is in keeping with **polymyositis**.

In **Lambert-Eaton Myasthenic Syndrome** you may see proximal muscle weakness associated with ocular muscle weakness and ptosis. It typically improves with repeated muscle use and is associated with small-cell lung cancer. It is not however associated with a raised CK.

Patients with **dermatomyositis** may present similarly but would also have skin changes such as a photosensitive facial rash and/or Gottron's papules which distinguishes the diagnosis from polymyositis.

In **Myasthenia Gravis** you would expect fatigable weakness, typically of the ocular muscles leading to double vision and ptosis. Proximal limb muscles, respiratory muscles and bulbar function may also be affected. However, you would not expect a raised CK.

In **polymyalgia rheumatica** patients may have proximal muscle pain, typically hip and shoulder pain, with fatigue and raised inflammatory markers. You would not expect to have a raised CK.



Next question >

# Polymyositis \*

#### Overview

- inflammatory disorder causing symmetrical, proximal muscle weakness
- thought to be a T-cell mediated cytotoxic process directed against muscle fibres
- may be idiopathic or associated with connective tissue disorders
- associated with malignancy
- dermatomyositis is a variant of the disease where skin manifestations are prominent, for example a purple (heliotrope) rash on the cheeks and eyelids
- typically affects middle-aged, female:male 3:1

## **Features**

- proximal muscle weakness +/- tenderness
- Raynaud's
- respiratory muscle weakness
- interstitial lung disease
  - o e.g. fibrosing alveolitis or organising pneumonia
  - o seen in around 20% of patients and indicates a poor prognosis
- dysphagia, dysphonia

# Investigations

- elevated creatine kinase
- other muscle enzymes (lactate dehydrogenase (LDH), aldolase, AST and ALT) are also elevated in 85-95% of patients
- EMG
- muscle biopsy
- anti-synthetase antibodies
  - anti-Jo-1 antibodies are seen in pattern of disease associated with lung involvement,
     Raynaud's and fever

# Management

- high-dose corticosteroids tapered as symptoms improve
- azathioprine may be used as a steroid-sparing agent



Next question >



# Textbooks High-yield textbook Extended textbook



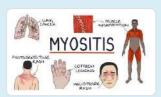
# Polymyositis and Dermatomyositis in 3 Minutes

Townsend Teaching - YouTube









Understanding Myositis (Polymyositis and Dermatomyositis)

Zero To Finals - YouTube







Report broken media

Score: 22.8%

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# Question 60 of 79





A 70-year-old man with rheumatoid arthritis presents for his monthly monitoring bloods because he is on methotrexate. He takes methotrexate 20mg once a week with folic Acid 5mg once weekly on a different day. He currently feels well in himself and his arthritis is well controlled. He does not drink alcohol or smoke. His monitoring blood tests come back as follows:

Hb	140 g/l	Na <sup>+</sup>	139 mmol/l	Bilirubin	14 µmol/l
Platelets	240 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.2 mmol/l	ALP	100 u/l
WBC	7 * 10 <sup>9</sup> /l	Urea	5 mmol/l	ALT	80 u/l
Neuts	4.5 * 10 <sup>9</sup> /l	Creatinine	87 µmol/l		

What is the correct course of action?

Stop methotrexate	
Reduce methotrexate dose to 10mg once weekly	
Switch methotrexate to sulfasalazine	
Continue on current dose with repeat bloods in one month	
Stop methotrexate and urgent liver USS	

Submit answer

Reference ranges ∨

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WBC	7 * 10 <sup>9</sup> /l	Urea	5 mmol/l	ALT	80 u/l
Neuts	4.5 * 10 <sup>9</sup> /l	Creatinine	87 µmol/l		

What is the correct course of action?



There is no need to stop methotrexate unless the alanine transaminase (ALT) or aspartate transaminase (AST) doubles according to the BSR guidelines. The patient should continue to have monthly blood test monitoring in the mean time.

See link below for full guidelines:

http://www.rheumatology.org.uk/includes/documents/cmdocs/2009/d/diseasemodifyingantirheumaticdrugdmardtheumaticdrugdmard



Next question >

# Methotrexate \*

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

#### Indications

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

#### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - o the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - o typically develops within a year of starting treatment, either acutely or subacutely
  - o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

#### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

## Prescribing methotrexate

- methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines
  recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until
  therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

#### Interactions

- avoid prescribing trimethoprim or co-trimoxazole concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

## Methotrexate toxicity

• the treatment of choice is folinic acid

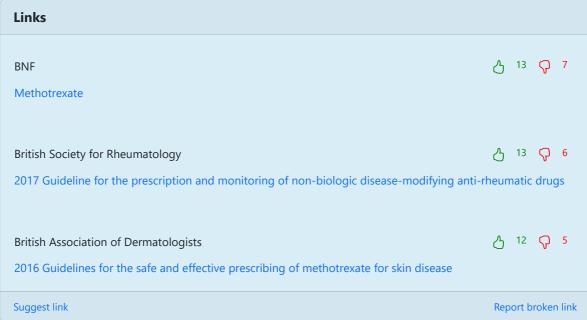


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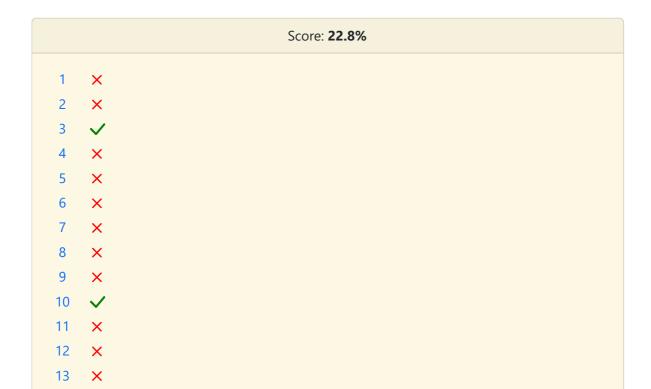


High-yield textbook

Extended textbook







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Question 61 of 79

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A 54-year-old patient is referred to rheumatology with reports of 12 months of dry eyes and dry mouth that she finds very troublesome. There are no other symptoms of note. She has a past medical history of coeliac disease and is compliant with a gluten-free diet.

On examination, she has dry oral mucosa. Chest auscultation is normal. There are is no nail fold vasculitis. There is no cervical, axillary or inguinal lymphadenopathy.

# Blood tests:

Hb	136 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10 <sup>9</sup> /L	(150 - 400)
WBC	5.2 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	137 mmol/L	(135 - 145)
K <sup>+</sup>	4.2 mmol/L	(3.5 - 5.0)
Urea	4.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
ESR	88 mm/Hr	(0-20)
Antinuclear antibody	positive (1:1280)	

What is the most definitive way of confirming the likely diagnosis?

Extractable nuclear antigen	
Measurement of salivary flow	
Salivary gland biopsy	
Schirmer's test	
Slit lamp examination	

Submit answer

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Question 61 of 79



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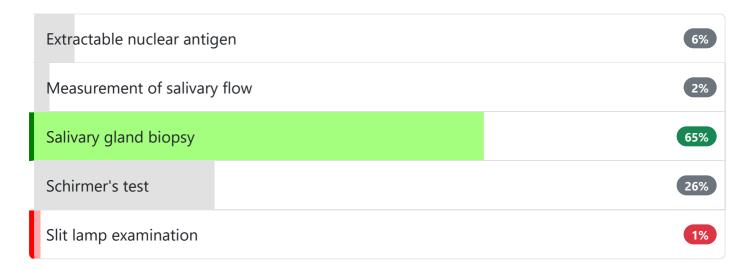
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Creatinine	88 µmol/L	(55 - 120)
CRP	4 mg/L	(< 5)
ESR	88 mm/Hr	(0-20)
Antinuclear antibody	positive (1:1280)	

What is the most definitive way of confirming the likely diagnosis?



Salivary gland biopsy is the most definitive way of confirming the diagnosis of primary Sjogren's syndrome - sections will show a typical lymphocytic infiltrate



**Salivary gland biopsy** is correct. This patient presents with sicca symptoms (dry eyes and dry mouth), raised ESR and a strongly positive antinuclear antibody. There are no symptoms of another connective tissue disease. This suggests a diagnosis of primary Sjogren's syndrome. The most definitive way of confirming the diagnosis is to do a salivary gland biopsy. This will show focal lymphocytic sialadenitis.

**Extractable nuclear antigen** is incorrect. Typically, if there is a positive antinuclear antibody result, then the next step is to try to subclassify the serology further by testing the ENA. Anti-Ro and anti-La antibodies are associated with Sjogren's syndrome and may support the diagnosis. However, similar to the other tests above, they are not as specific for the diagnosis as a lip biopsy and therefore not as definitive.

**Measurement of salivary flow** is incorrect. A whole saliva unstimulated flow rate of <= 0.1mL/minute suggests salivary gland hypofunction and can be used to aid a diagnosis of Sjogren's syndrome. However, it is not as specific for the diagnosis as a positive salivary gland biopsy.

**Schirmer's test** is incorrect. This can be used to aid the diagnosis of Sjogren's syndrome. It involves placing a strip of filter paper inside the lower eyelid. The eyes are shut for 5 minutes. The paper is removed and the amount of moisture is calculated. Less than 5mm in 5 minutes is considered an abnormal result. It is not as specific for the diagnosis of primary Sjogren's syndrome as proving focal lymphocytic sialadenitis pathologically.

**Slit lamp examination** is incorrect. An ophthalmology review may elicit severe dryness and ocular inflammation. This may suggest Sjogren's syndrome. However, there will be other differentials that can cause these features and a biopsy is more specific for the diagnosis.



Next question >

# Sjogren's syndrome

Sjogren's syndrome is an autoimmune disorder affecting exocrine glands resulting in dry mucosal surfaces. It may be primary (PSS) or secondary to rheumatoid arthritis or other connective tissue disorders, where it usually develops around 10 years after the initial onset. Sjogren's syndrome is much more common in females (ratio 9:1). There is a marked increased risk of lymphoid malignancy (40-60 fold).

# **Features**

- dry eyes: keratoconjunctivitis sicca
- dry mouth

- vaginal dryness
- arthralgia
- Raynaud's, myalgia
- sensory polyneuropathy
- recurrent episodes of parotitis
- renal tubular acidosis (usually subclinical)

# Investigation

- rheumatoid factor (RF) positive in nearly 50% of patients
- ANA positive in 70%
- anti-Ro (SSA) antibodies in 70% of patients with PSS
- anti-La (SSB) antibodies in 30% of patients with PSS
- Schirmer's test: filter paper near conjunctival sac to measure tear formation
- histology: focal lymphocytic infiltration
- also: hypergammaglobulinaemia, low C4

# Management

- artificial saliva and tears
- pilocarpine may be helpful to stimulate saliva production



Next question >





Media



# Sjogren's syndrome

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6

A 47-year-old woman was admitted with a 24-hour history of acute breathlessness. She had a three-month history of progressive weakness, 8 kg weight loss, and progressive dysphagia. She was a non-smoker. On examination, her temperature was 38.4°C, pulse 96 beats per minute, and blood pressure 136/65 mmHg. Auscultation revealed coarse inspiratory crackles at the right base and mid zone. She had grade 4/5 weakness of the proximal muscles of the upper and lower limbs. There were no other abnormalities.

### Investigations:

Hb	130g/l (115-165)
Platelets	238 * 10 <sup>9</sup> /l (150-400)
WBC	16 * 10 <sup>9</sup> /l (4.0-11.0)
CRP	130mg/l (<10)
Na <sup>+</sup>	140mmol/l (135-145)
K <sup>+</sup>	3.7mmol/l (3.5-4.5)
Urea	7.5mmol/l (5.0-9.0)
Creatinine	98µmol/l (60-11)
Creatine kinase	5230U/I (24-170)

Which of the following is most likely to be helpful in making a specific diagnosis?

Anti-double-stranded DNA antibodies	
Anti-synthetase antibodies	
Anti-nuclear antibodies	
Anti-smooth muscle antibodies	
Rheumatoid factor	

Submit answer

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Question 62 of 79

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 $\Box$ 

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Urea	7.5mmol/l (5.0-9.0)
Creatinine	98μmol/l (60-11)
Creatine kinase	5230U/I (24-170)

Which of the following is most likely to be helpful in making a specific diagnosis?

Anti-double-stranded DNA antibodies	5%
Anti-synthetase antibodies	73%
Anti-nuclear antibodies	11%
Anti-smooth muscle antibodies	11%
Rheumatoid factor	1%

Anti-synthetase antibodies are specifically associated with polymyositis

Important for me Less important



The diagnosis is in this scenario is aspiration pneumonia secondary to dysphagia caused by polymyositis. It is an idiopathic inflammatory myopathy characterized by symmetrical, proximal muscle weakness and elevated skeletal muscle enzyme levels (creatine kinase). A number of autoantibodies are seen with polymyositis; however, many of these are not disease-specific. Rheumatoid factor is seen in at least 50% of cases of polymyositis, whilst anti-nuclear antibodies (ANA) are seen in approximately 30% of cases. Anti-double-stranded DNA antibodies are more specific for systemic lupus erythematosus (SLE). Anti-smooth muscle antibodies are seen in autoimmune hepatitis. Antibodies in myositis can be divided into those which are specific for myositis, and those which are associated with myositis.

Myositis-specific antibodies are targeted against three types of proteins:

- Transfer ribonucleic acid (tRNA) synthetases: whilst a number of antibodies have been described, the main one is the Anti-Jo-1 antibody. Patients who are Anti-Jo-1 positive tend to have interstitial lung disease, arthritis (non-deforming) and fevers.
- Nuclear Mi-2 protein: these are seen in approximately 20% of patients with myositis, and are more specific for dermatomyositis.
- Signal recognition peptide (SRP): anti-SRP antibodies are seen in approximately 5% of
  patients with polymyositis, and are associated with poor response to treatment and a poor
  prognosis.

Myositis-associated antibodies are seen in up to 50% of patients with myositis. They are typically seen in other connective tissue diseases. Examples include anti-PM/Scl antibodies (seen in polymyositis/systemic sclerosis overlap disease), and anti-Ku antibodies, seen in overlap myositis/connective tissue diseases



Next question >

### Polymyositis \*

### Overview

- inflammatory disorder causing symmetrical, proximal muscle weakness
- thought to be a T-cell mediated cytotoxic process directed against muscle fibres
- may be idiopathic or associated with connective tissue disorders
- associated with malignancy
- dermatomyositis is a variant of the disease where skin manifestations are prominent, for example a purple (heliotrope) rash on the cheeks and eyelids
- typically affects middle-aged, female:male 3:1

### **Features**

proximal muscle weakness +/- tenderness

- Raynaud's
- respiratory muscle weakness
- interstitial lung disease
  - o e.g. fibrosing alveolitis or organising pneumonia
  - o seen in around 20% of patients and indicates a poor prognosis
- dysphagia, dysphonia

### Investigations

- elevated creatine kinase
- other muscle enzymes (lactate dehydrogenase (LDH), aldolase, AST and ALT) are also elevated in 85-95% of patients
- EMG
- muscle biopsy
- anti-synthetase antibodies

+ Q 123

anti-Jo-1 antibodies are seen in pattern of disease associated with lung involvement,
 Raynaud's and fever

### Management

- high-dose corticosteroids tapered as symptoms improve
- azathioprine may be used as a steroid-sparing agent





### Media



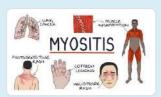
### Polymyositis and Dermatomyositis in 3 Minutes

Townsend Teaching - YouTube









Understanding Myositis (Polymyositis and Dermatomyositis)

Zero To Finals - YouTube







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Score: 22.8%

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Question 63 of 79





A 71-year-old man is being treated for urosepsis. For the past seven days, he has experienced significant back pain which is exacerbated when mobilising with the physiotherapy team. He has occasionally suffered from back pain in the past however reports that his pain at present is far more severe than anything he is used to.

He has a past medical history of type 2 diabetes and adenocarcinoma of the colon for which he is awaiting definitive surgical treatment.

An MRI spine (T2) is performed and displayed below:



© Image used on license from Radiopaedia



### What is the most likely diagnosis?

Bony metastases	
Disc prolapse	
Discitis	

Osteoarthritis	
Vertebral fracture	

### Submit answer

Reference ranges ✓

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Question 63 of 79

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A 71-year-old man is being treated for urosepsis. For the past seven days, he has experienced significant back pain which is exacerbated when mobilising with the physiotherapy team. He has occasionally suffered from back pain in the past however reports that his pain at present is far more severe than anything he is used to.

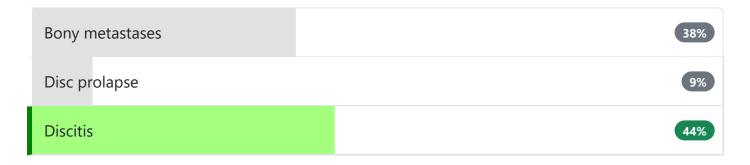
He has a past medical history of type 2 diabetes and adenocarcinoma of the colon for which he is awaiting definitive surgical treatment.

An MRI spine (T2) is performed and displayed below:



© Image used on license from Radiopaedia

What is the most likely diagnosis?



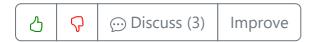
This man's MRI shows a high signal intensity mass within the T10-11 disc space which is indicative of discitis. He is at a significantly increased risk of developing this condition given his ongoing systemic infection and immunocompromise (type 2 diabetes and active cancer).

Bony metastases would be of particular concern given this man's adenocarcinoma of the colon however the MRI images do not show any bony pathology. Instead, the radiological abnormality relates to the T10-11 intervertebral disc. Metastases are therefore an incorrect answer.

Disc prolapse is a differential diagnosis here given this man's ongoing physiotherapy. The mild impingement of the spinal cord from the intervertebral disc here is in keeping with prolapse however the increased intensity signal and inflammation is characteristic of discitis which is, therefore, the correct answer.

Due to the acute onset of symptoms and the MRI indicative of discitis, osteoarthritis of the spine is not the most likely diagnosis here.

The MRI shows an intervertebral disc pathology but no vertebral fractures. This is therefore also incorrect.



Next question >

### Discitis \*

Discitis is an infection in the intervertebral disc space. It can lead to serious complications such as sepsis or an epidural abscess.

### **Features**

- Back pain
- General features
  - o pyrexia,
  - o rigors
  - o sepsis
- Neurological features
  - e.g. changing lower limb neurology
  - if an epidural abscess develops

### Causes

- Bacterial
  - o Staphylococcus aureus is the most common cause of discitis
- Vira
- TB
- Aseptic

### Diagnosis

- Imaging: MRI has the highest sensitivity
- CT-guided biopsy may be required to guide antimicrobial treatment



### **Treatment**

- The standard therapy requires six to eight weeks of intravenous antibiotic therapy
- Choice of antibiotic is dependent on a variety of factors. The most important factor is to identify the organism with a positive culture (e.g. blood culture, or CT-guided biopsy)
- the patient should be assessed for endocarditis e.g. with transthoracic echo or transesophageal echo. Discitis is usually due to haematogenous seeding of the vertebrae implying that the patient has had a bacteraemia and seeding could have occurred elsewhere

### Complications

- sepsis
- epidural abscess



Next question >



## Textbooks High-yield textbook Extended textbook

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Question 64 of 79





A 43-year-old patient presents to the emergency department with one day of fever, abdominal pain, joint pain and chest pain. This is the third time this has occurred. He has no past medical history and does not take any regular medications. He is of Turkish descent.

### Observations:

- Temperature 38.4
- Blood pressure 120/66 mmHg
- Respiratory rate 18/minute
- Oxygen saturations 96% on room air
- Heart rate 88 beats per minute

On examination, there is generalized abdominal tenderness. Synovitis is detectable at the left ankle. There is a pericardial rub.

### Blood tests:

Hb	138 g/L	Male: (135-180) Female: (115 - 160)
Platelets	189 * 10 <sup>9</sup> /L	(150 - 400)
WBC	4.5 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Na <sup>+</sup>	137 mmol/L	(135 - 145)
K <sup>+</sup>	4.2 mmol/L	(3.5 - 5.0)
Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	52 mg/L	(< 5)
Antinuclear antibody	negative	(negative)

What is the most appropriate medication to administer?

Anakinra	
Co-amoxiclav	
Colchicine	
Hydroxychloroquine	

### Submit answer

Reference ranges  $\checkmark$ 

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Question 64 of 79



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Urea	5.2 mmol/L	(2.0 - 7.0)
Creatinine	88 µmol/L	(55 - 120)
CRP	52 mg/L	(< 5)
Antinuclear antibody	negative	(negative)

What is the most appropriate medication to administer?



### Colchicine may be helpful in Familial Mediterranean Fever

Important for me Less important

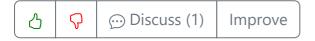
**Colchicine** is the correct answer. This patient presents with recurrent episodes of fever, abdominal pain, chest pain and joint pain. Clinical examination suggests arthritis, peritonitis and pericarditis. Additionally, he is of Turkish descent. Taken together, this suggests a diagnosis of Familial Mediterranean Fever (FMF). The first-line treatment of FMF is colchicine. It can abort and prevent attacks.

**Anakinra** is incorrect. This is an IL-1 inhibitor biologic medication. There is some literature to suggest this can be effective in refractory FMF but it is not first-line therapy.

**Co-amoxiclav** is incorrect. The recurrent constellation of arthritis, pericarditis, peritonitis and fever is more suggestive of FMF than an infection.

**Hydroxychloroquine** is incorrect. This medication can be used to treat systemic lupus erythematosus. While SLE can cause pericarditis, arthritis, fever and in rare cases, peritonitis, the antinuclear antibody is negative, making this diagnosis very unlikely.

**Prednisolone** is incorrect. This can be used to treat protracted febrile myalgia in FMF but is not first-line therapy.



Next question >

### Familial Mediterranean Fever \*

Familial Mediterranean Fever (FMF, also known as recurrent polyserositis) is an autosomal recessive disorder which typically presents by the second decade. It is more common in people of Turkish, Armenian and Arabic descent.

Features - attacks typically last 1-3 days

- pyrexia
- abdominal pain (due to peritonitis)
- pleurisy
- pericarditis
- arthritis
- erysipeloid rash on lower limbs

### Management

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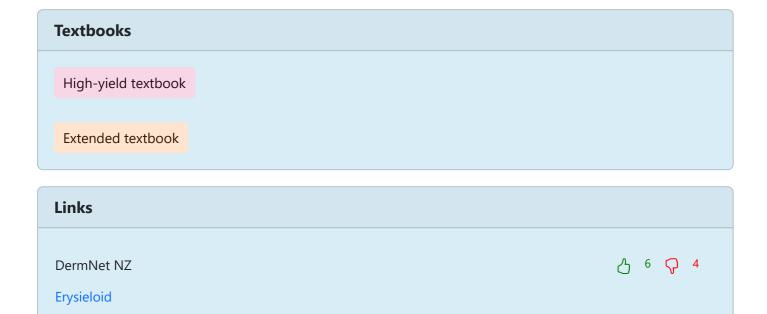
• colchicine may help

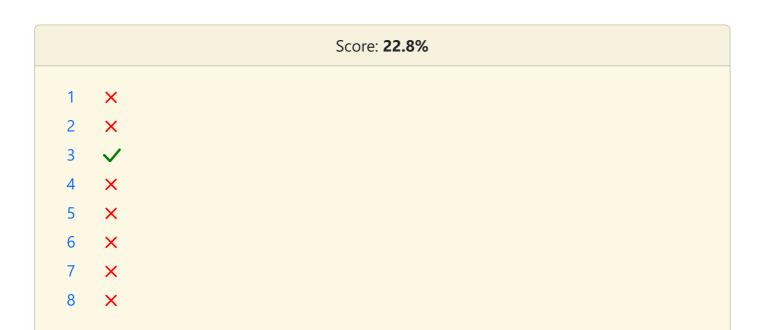


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A 55-year-old woman presents for review. Her mother has just been discharged after suffering a hip fracture. She is concerned that she may have 'inherited' osteoporosis and is asking what she should do. She has no significant past medical history of note, takes no regular medication and has never sustained any fractures. She smokes around 20 cigarettes per day and drinks about 3-4 units of alcohol per day.

What is the most appropriate course of action?

Arrange bone mineral density measurement (DEXA scan)	
Reassure her that assessment of fragility fracture risk does not need to be years	done until 65
Refer her to the genetics team for a risk assessment	
Start first-line bone protection (i.e. ensure calcium/vitamin D replete + orabisphosphonate)	al ×
Use the FRAX tool	

Submit answer

Reference ranges ∨

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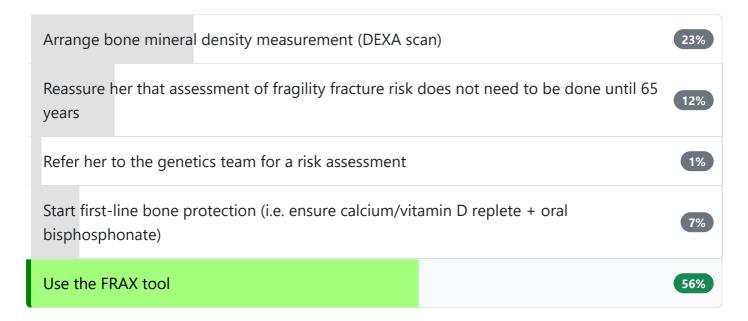


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What is the most appropriate course of action?



This lady has a number of risk factors for developing osteoporosis:

- positive family history
- smoking
- excess alcohol intake

She should therefore have an immediate FRAX assessment, rather than waiting until 65 years as we would for women without any relevant risk factors



Next question >

### Osteoporosis: assessing risk \*

We worry about osteoporosis because of the increased risk of fragility fractures. So how do we assess which patients are at risk and need further investigation?

NICE produced guidelines in 2012: Osteoporosis: assessing the risk of fragility fracture. The following is based on those guidelines.

They advise that all women aged >= 65 years and all men aged >= 75 years should be assessed. Younger patients should be assessed in the presence of risk factors, such as:

- previous fragility fracture
- current use or frequent recent use of oral or systemic glucocorticoid
- history of falls
- family history of hip fracture
- other causes of secondary osteoporosis, for example:
  - hypogonadism in either sex including low testosterone in men and premature menopause in women
  - o endocrine conditions, including diabetes mellitus, Cushing's disease, hyperthyroidism
  - o conditions associated with malabsorption, including inflammatory bowel disease, coeliac disease, and chronic pancreatitis.
  - o rheumatoid arthritis and other inflammatory arthropathies.
- low body mass index (BMI) (less than 18.5 kg/m²)
- smoking
- alcohol intake of more than 14 units per week for women and more than 14 units per week for men.

### Methods of risk assessment

The first step is to exclude secondary causes of osteoporosis as underlying conditions may also require treatment. Examples of secondary causes are listed above.

If a patient has had a recent fragility fracture, non-osteoporotic causes should be looked for, for example, bone metastases, myeloma and Paget's disease.

A DEXA scan should be offered without calculating the fragilty risk score in the following situations:

- > 50 years of age with a history of fragility fracture
- < 40 years of age who have a major risk factor for fragility fracture these patients should be referred to a specialist depending on the T-score
- before starting treatments that may have a rapid adverse effect on bone density (for example, sex hormone deprivation for treatment for breast or prostate cancer)

### Fragility fracture risk score

NICE recommends using a clinical prediction tool such as FRAX or QFracture to assess a patient's 10-year risk of developing a fracture. These take into account a number of factors, including the risk factors listed above. This is analogous to the cardiovascular risk tools such as QRISK.

### Interpreting the results of FRAX

It is important to note though that there is a lot of room for pragmatism and clinical judgement in these guidelines. This reflects the approximate nature of risk scoring and multiple factors that determine fracture risk.

### **OFracture**

• if the 10-year fracture risk is ≥ 10% then a DEXA scan should be arranged

### **FRAX**

- a colour 'risk' is given by the calculator green, orange or red
- patients in the orange zone should have a DEXA scan if not already done to further refine their 10-year risk
- patients in the red zone should also have a DEXA scan if not already done to act as a baseline and guide drug treatment

### When should we reassess a patient's risk?

NICE recommend that we recalculate a patient's risk (i.e. repeat the FRAX/QFracture):

- if the original calculated risk was in the region of the intervention threshold for a proposed treatment and only after a minimum of 2 years, or
- when there has been a change in the person's risk factors



Next question >



### **Textbooks**

High-yield textbook

Extended textbook

Links	
NICE 2012 Osteoporosis: assessing the risk of fragility fracture	<b>△</b> 8 <b>♀</b> 10
Royal College of Physicians  2012 Recent advances in the risk assessment and treatment of osteoporosis	<b>占</b> 10 <b>Ç</b> 7
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6

Question 66 of 79

P

 $\Rightarrow$ 

A 79-year-old woman complains of pain in her hands. An x-ray is ordered:



© Image used on license from Radionaedia



Based on the x-ray findings, what is the most likely diagnosis?

Gout	
Primary hyperparathyroidism	
Rheumatoid arthritis	
Osteoarthritis	
Paget's disease	

Submit answer

Reference ranges ✓

## Score: 0%

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6

Question 66 of 79

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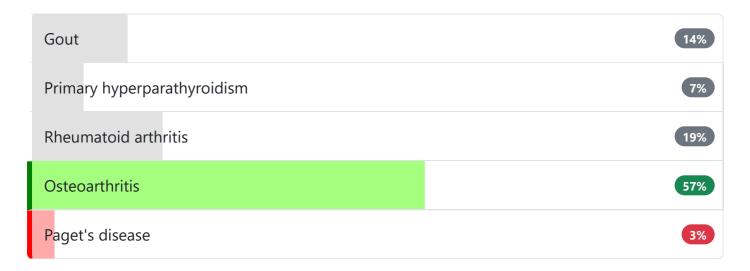
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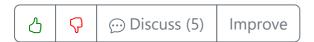
A 79-year-old woman complains of pain in her hands. An x-ray is ordered:



Based on the x-ray findings, what is the most likely diagnosis?



The distribution of joint problems (mainly distal interphalangeal joints and carpometacarpal joints) and changes seen (loss of joint space, subchondral sclerosis) points to a diagnosis of osteoarthritis.



# Osteoarthritis: x-ray changes \*

X-ray changes of osteoarthritis (LOSS)

- Loss of joint space
- Osteophytes forming at joint margins
- **S**ubchondral sclerosis
- **S**ubchondral cysts



Next question >







Score: **22.8%** 

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Question 67 of 79





A 58-year-old man presents with malaise and fever. He states that he has been feeling unwell for several months. His main complaints are intermittent swelling and pain of his right ear, painful red eyes, and arthralgia. He has no past medical history and takes no regular medicines.

On examination, you note right auricular swelling, bilateral anterior uveitis, and a symmetrical small joint polyarthritis.

Hb	115 g/l	Na <sup>+</sup>	138 mmol/l
Platelets	330 * 10 <sup>9</sup> /l	K <sup>+</sup>	4.2 mmol/l
WBC	13.1 * 10 <sup>9</sup> /l	Urea	6.2 mmol/l
Neuts	10.4 * 10 <sup>9</sup> /l	Creatinine	95 µmol/l
Lymphs	2.5 * 10 <sup>9</sup> /l	CRP	132 mg/l
Eosin	0.6 * 10 <sup>9</sup> /l	pANCA	negative
C3	normal	C4	normal
ANA	negative	Anti Sm	negative
RhF	positive	Anti CCP	negative

What is the most likely diagnosis?

Eosinophilic granulomatosis with polyangiitis	
Granulomatosis with polyangiitis	
Systemic lupus erythematosus	
Rheumatoid arthritis	
Relapsing polychondritis	

Submit answer

Reference ranges V

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Question 67 of 79



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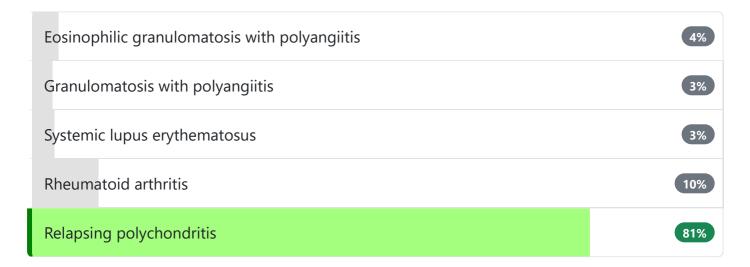


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Lymphs	2.5 * 10 <sup>9</sup> /l	CRP	132 mg/l
Eosin	0.6 * 10 <sup>9</sup> /l	pANCA	negative
C3	normal	C4	normal
ANA	negative	Anti Sm	negative
RhF	positive	Anti CCP	negative

What is the most likely diagnosis?



Relapsing polychondritis is a multi-systemic condition which most commonly causes relapsing episodes of auricular chondritis

Important for me Less important

Relapsing polychondritis is a multi-systemic condition characterised by repeated episodes of inflammation and deterioration of cartilage. It is often a painful disease which can cause joint deformity and be life-threatening if the respiratory tract, heart valves, or blood vessels are

affected. The exact mechanism is poorly understood, but it is thought to be related to an immune-mediated attack on cartilage proteins.

The history in this case is suggestive of relapsing polychondritis, however the clinical features are non-specific and can occur due to many of the other options. The laboratory date is therefore of great use in narrowing down the differential diagnosis.

A normal C3 and C4, and negative ANA, make SLE unlikely.

A negative pANCA and cANCA makes granulomatosis with polyangitis and eosinophilic granulomatosis less likely.

A negative anti-CCP makes rheumatoid arthritis less likely. Although RhF is positive, this is very non-specific and is positive in many other diseases and in the healthy general population.



Next question >

# Relapsing polychondritis \*

Relapsing polychondritis is a multi-systemic condition characterised by repeated episodes of inflammation and deterioration of cartilage. This most commonly affects the ears, however, can affect other parts of the body such as the nose and joints.

## Key features:

- Ears: auricular chondritis, hearing loss, vertigo
- Nasal: nasal chondritis → saddle-nose deformity
- Respiratory tract: e.g. hoarseness, aphonia, wheezing, inspiratory stridor
- Ocular: episcleritis, scleritis, iritis, and keratoconjunctivitis sicca
- Joints: arthralgia
- Less commonly: cardiac valcular regurgitation, cranial nerve palsies, peripheral neuropathies, renal dysfunction

## Diagnosis:

• Various scoring systems based on clinical, pathological, and radiological criteria

#### **Treatment**

- Induce remission: steroids
- Maintenance: azathioprine, methotrexate, cyclosporin, cyclophosphamide



Next question >

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Textbooks

High-yield textbook

Extended textbook

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## Question 68 of 79





A 54-year-old man presents to Gastroenterology outpatient clinic, for review of his Crohn's disease which was diagnosed 3 years ago. His other past medical history includes ischaemic heart disease, hypercholesterolaemia and gout. He has been suffering from a few months of increased diarrhoea and abdominal pain, and you feel he would benefit from starting azathioprine. Which medication is it important to ensure he is not taking before commencing azathioprine?

Ramipril	
Allopurinol	
Losartan	
Aspirin	
Simvastatin	

Submit answer

Reference ranges ∨

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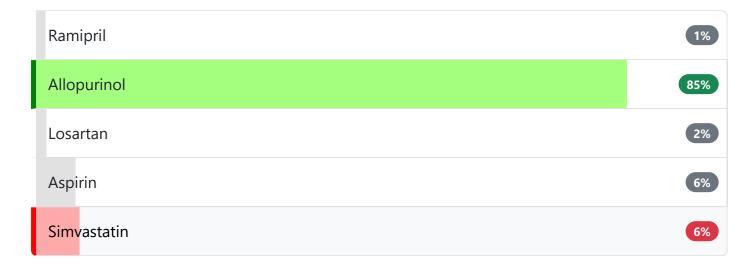
Question 68 of 79



 $\square$ 



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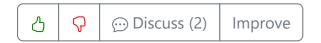


Azathioprine and allopurinol have a severe interaction causing bone marrow suppression

| Important for me | Less important |

Allopurinol inhibits the enzyme xanthine oxidase (XO). This enzyme is also required to inactive 6-mercaptopurine (the active agent from azathioprine). When XO is inhibited, this causes an increase in 6-mercaptopurine levels, which are then shunted down a different metabolic pathway, leading to higher levels of 6-thioguanine metabolites. There are incorporated into the DNA of white blood cells, leading to reduced activation and reduced replication potential.

Coadministration of azathioprine and allopurinol requires dose reductions and extra monitoring for life threatening agranulocytosis. The other medications listed do not interact with azathioprine and require no extra monitoring.



Next question >

# Azathioprine \*

Azathioprine is metabolised to the active compound mercaptopurine, a purine analogue that inhibits purine synthesis. A thiopurine methyltransferase (TPMT) test may be needed to look for

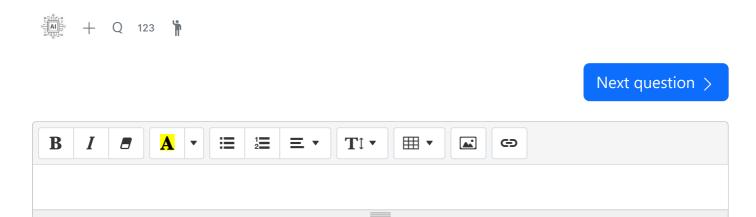
individuals prone to azathioprine toxicity.

Adverse effects include

- bone marrow depression
  - o consider a full blood count if infection/bleeding occurs
- nausea/vomiting
- pancreatitis
- increased risk of non-melanoma skin cancer

A significant interaction may occur with allopurinol and hence lower doses of azathioprine should be used.

Azathioprine is generally considered safe to use in pregnancy.







## Score: **22.8%**

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7	77	<b>✓</b>
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Question 69 of 79





A 45-year-old gentleman presents to the emergency department with proximal muscle weakness of increasing intensity over the last few days. Initially he put it down to restarting gym exercises, however this is progressively getting worse.

On examination you note a slim gentleman who has difficulty in rising from the chair, and that appears to be in pain on examination of the deltoids and quadriceps. He has low grade pyrexia (37.3), however he exhibits no rashes or evidence of photosensitivity.

His bloods are as follow:

Hb	134 g/l	Na <sup>+</sup>	140 mmol/l	Bilirubin	18 µmol/l
Platelets	355 * 10 <sup>9</sup> /l	K <sup>+</sup>	3.7 mmol/l	ALP	86 u/l
WBC	9.3 * 10 <sup>9</sup> /l	Urea	7.7 mmol/l	ALT	101 u/l
Neuts	5.5 * 10 <sup>9</sup> /l	Creatinine	87 µmol/l	γGT	110 u/l
Lymphs	3.3 * 10 <sup>9</sup> /l			Albumin	32 g/l
Eosin	0.14 * 10 <sup>9</sup> /l			CRP	125 mg/l
СК	2158 IU/L				

Which investigations will confirm this patient's diagnosis:

Anti-Jo1 antibody screen	
Electromyography	
Ultrasound of muscles affected	
Muscle biopsy	
Magnetic Resonance Imaging	

Submit answer

Reference ranges  $\vee$ 

## Score: 0%

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Question 69 of 79





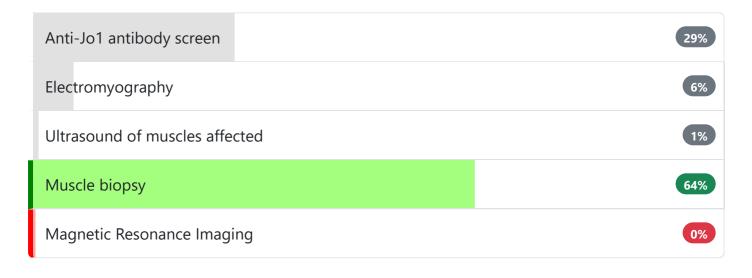
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Platelets	355 * 10 <sup>9</sup> /l	K <sup>+</sup>	3.7 mmol/l	ALP	86 u/l
WBC	9.3 * 10 <sup>9</sup> /l	Urea	7.7 mmol/l	ALT	101 u/l
Neuts	5.5 * 10 <sup>9</sup> /l	Creatinine	87 µmol/l	γGT	110 u/l
Lymphs	3.3 * 10 <sup>9</sup> /I			Albumin	32 g/l
Eosin	0.14 * 10 <sup>9</sup> /l			CRP	125 mg/l
СК	2158 IU/L				

Which investigations will confirm this patient's diagnosis:



Muscle biopsy is the gold standard investigation for the diagnosis of polymyositis

Important for me Less important

The history and examination findings, alongside the rise in Creatinine Kinase in this gentleman should alert us towards the potential diagnosis of Polymyositis. All of the options provided above are useful in the diagnosis of polymyositis, which makes this question slightly tricky. Anti-Jo-1 antibodies are characteristically associated with polymyositis and indeed they are likely to point us to the right direction; however given the fact that it is an Anti-nuclear antibody subtype it can be raised in other systemic inflammatory condition such as Systemic Lupus Erythematosus and Mixed Connective Tissue disorder. Electromyography can be used to distinguish between myopathic and neuropathic causes of weakness and isolate the muscle group involved; however it is fairly non-specific. Magnetic Resonance Imaging (MRI) is nowadays commonly employed, having the benefit of being non-invasive and able to display large muscle groups, it has a high sensitivity however it's non specific and cannot necessarily differentiate the changes of inflammatory myopathy. Ultrasound of muscles has now been surpassed by the use of MRI; this leaves us with muscle biopsy. Even though it is less commonly used today due to its invasive nature and difficulty in accessing the appropriate muscle group, it remains the most sensitive and specific test in identifying polymyositis histopathologically, provided that the right area of muscle is biopsied.



Next question >

## Polymyositis \*

#### Overview

- inflammatory disorder causing symmetrical, proximal muscle weakness
- thought to be a T-cell mediated cytotoxic process directed against muscle fibres
- may be idiopathic or associated with connective tissue disorders
- associated with malignancy
- dermatomyositis is a variant of the disease where skin manifestations are prominent, for example a purple (heliotrope) rash on the cheeks and eyelids
- typically affects middle-aged, female:male 3:1

#### **Features**

- proximal muscle weakness +/- tenderness
- Raynaud's
- respiratory muscle weakness
- interstitial lung disease
  - o e.g. fibrosing alveolitis or organising pneumonia
  - seen in around 20% of patients and indicates a poor prognosis
- dysphagia, dysphonia

## Investigations

- elevated creatine kinase
- other muscle enzymes (lactate dehydrogenase (LDH), aldolase, AST and ALT) are also elevated in 85-95% of patients
- EMG
- muscle biopsy

- anti-synthetase antibodies
  - anti-Jo-1 antibodies are seen in pattern of disease associated with lung involvement,
     Raynaud's and fever

## Management

- high-dose corticosteroids tapered as symptoms improve
- azathioprine may be used as a steroid-sparing agent



Next question >



## **Textbooks**

High-yield textbook

Extended textbook

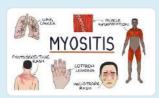
#### Media



Polymyositis and Dermatomyositis in 3 Minutes

Townsend Teaching - YouTube





Understanding Myositis (Polymyositis and Dermatomyositis)

Zero To Finals - YouTube



## Score: **22.8%**

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A 41-year-old woman presents with a lump over the wrist. It has been present for approximately 6 months and is not painful. She does not experience any symptoms from it and has no significant past medical history. On examination, the lump appears as shown below:



Given the likely diagnosis, what is the most appropriate next step?

Aspiration	
Reassure and monitor	
Surgical referral	
Ultrasound	
Wrist X-ray	

Submit answer

Reference ranges  $\vee$ 

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Question 70 of 79



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A 41-year-old woman presents with a lump over the wrist. It has been present for approximately 6 months and is not painful. She does not experience any symptoms from it and has no significant past medical history. On examination, the lump appears as shown below:



Given the likely diagnosis, what is the most appropriate next step?



This appearance of this lesion is highly suggestive of a ganglion. These are simple cysts and are the most common benign lesions of the hand and wrist. Typically, they appear as seen here, over the dorsal aspect of the wrist. Onset is usually insidious. They often cause no symptoms and patients can simply be **reassured and advised to monitor**.

Aspiration is incorrect. This can be both diagnostic, if uncertain, and therapeutic. In this case,

there is no indication for aspiration as the patient is not experiencing any symptoms.

A **surgical referral** is unwarranted here. This is typically employed when symptoms are severe or there are neurovascular manifestations.

**Ultrasound** scanning is not necessary here. The photo very clearly shows a ganglion cyst and the patient is not experiencing any symptoms.

Similarly, **X-ray** is unwarranted. The photo, and history, allow us to be almost certain of the diagnosis and so imaging is not required.



Next question >

## Ganglion \*

A ganglion presents as a 'cyst' arising from a joint or tendon sheath. They are most commonly seen around the dorsal aspect of the wrist and are 3 times more common in women.

## **Features**

• a firm and well-circumscribed mass that transilluminates

## Management

- ganglions often disappear spontaneously after several months
- surgical excision is indicated for cysts associated with severe symptoms or neurovascular manifestations



Next question >



## **Textbooks**

High-yield textbook

Extended textbook

## Score: **22.8%** X 1 X 2 **✓** 3 X 4 5 X X 6 X 7 X 8 × 9 10 X 11 X 12 × 13 × 14 × 15 × 16 17 🗸 × 18 **✓** 19 20 X X 21 22 X 23 X 24 X 25 🗸 26 🗸 27 X 28 X × 29

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## Question 71 of 79

P

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A 46-year-old lady complains of proximal muscle weakness over 6 months. History is otherwise unremarkable. She has no past medical history and is on no medications. She does not drink alcohol. On examination, power is 4/5 proximally in both arms and legs, otherwise unremarkable. Blood investigations are normal except for an elevated creatinine kinase level at 900 U/L. Electromyography demonstrates myopathic features. You order a muscle biopsy to help differentiate which myopathy is present. The results show endomysial lymphocytic infiltrates that invade nonnecrotic muscle fibres. What is the most likely diagnosis?

Dermatomyositis	
Inclusion body myositis	
Systemic lupus erythromatosis	
Lung cancer	
Polymyositis	

Submit answer

Reference ranges  $\vee$ 

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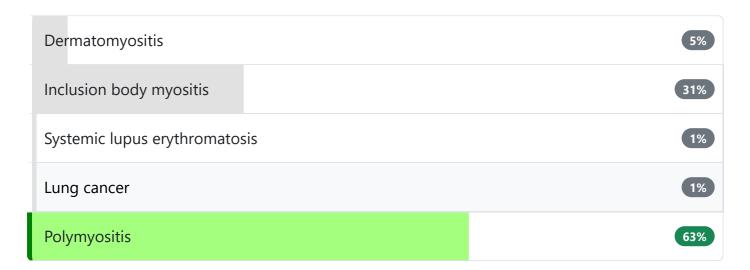
Question 71 of 79



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A 46-year-old lady complains of proximal muscle weakness over 6 months. History is otherwise unremarkable. She has no past medical history and is on no medications. She does not drink alcohol. On examination, power is 4/5 proximally in both arms and legs, otherwise unremarkable. Blood investigations are normal except for an elevated creatinine kinase level at 900 U/L. Electromyography demonstrates myopathic features. You order a muscle biopsy to help differentiate which myopathy is present. The results show endomysial lymphocytic infiltrates that invade nonnecrotic muscle fibres. What is the most likely diagnosis?



Proximal muscle weakness is a nonspecific complaint and the elevated CK with this presentation suggests a myopathy, of which the common are inflammatory (polymyositis, dermatomyositis, and inclusion body myositis), toxic myopathies (e.g. statin- or alcohol-induced), and inherited (Duchenne/Becker muscular dystrophy, myotonic dystrophy). There is no history of toxic agents here and the history is not lifelong to suggest an inherited causes. There are no skin changes to suggest dermatomyositis. The diagnostic difficulty may be between inclusion body myositis and polymyositis. The former tends to affect wrists and fingers more than the latter. Where there is diagnostic difficulty clinically, a biopsy may help make a diagnosis:

Endomysial lymphocytic infiltrates that invade nonnecrotic muscle fibres = polymyositis

Perimysial inflammation of lymphocytes and parafascicular atrophy = dermatomyositis

Inflammatory infiltrates and inclusions within muscle fibres = inclusion body myositis.

Polymyositis responds to immunosuppression.

It is likely she has a polymyositis and coincidental carpal tunnel syndrome rather than an inclusion body myositis.

## Polymyositis \*

### Overview

- inflammatory disorder causing symmetrical, proximal muscle weakness
- thought to be a T-cell mediated cytotoxic process directed against muscle fibres
- may be idiopathic or associated with connective tissue disorders

**Improve** 

- associated with malignancy
- dermatomyositis is a variant of the disease where skin manifestations are prominent, for example a purple (heliotrope) rash on the cheeks and eyelids
- typically affects middle-aged, female:male 3:1

## **Features**

- proximal muscle weakness +/- tenderness
- Raynaud's
- respiratory muscle weakness
- interstitial lung disease
  - o e.g. fibrosing alveolitis or organising pneumonia
  - o seen in around 20% of patients and indicates a poor prognosis
- dysphagia, dysphonia

## Investigations

- elevated creatine kinase
- other muscle enzymes (lactate dehydrogenase (LDH), aldolase, AST and ALT) are also elevated in 85-95% of patients
- EMG
- muscle biopsy
- anti-synthetase antibodies
  - anti-Jo-1 antibodies are seen in pattern of disease associated with lung involvement,
     Raynaud's and fever

## Management

- high-dose corticosteroids tapered as symptoms improve
- azathioprine may be used as a steroid-sparing agent





## **Textbooks**

High-yield textbook

Extended textbook

## Media



Polymyositis and Dermatomyositis in 3 Minutes

Townsend Teaching - YouTube





Understanding Myositis (Polymyositis and Dermatomyositis)

Zero To Finals - YouTube



Report broken media

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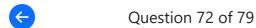
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9. A 39 year old woman presents to her GP with symptoms of dysuria and increased urinary frequency for the past three days. She also complains of lower abdominal pain but has no overt signs of systemic sepsis. Examination is entirely normal aside from mild suprapubic pain. Urine dip correlates with a diagnosis of urinary tract infection with positive nitrites, leukocytes, blood and protein. The sample is sent for culture. The patients medical history is significant only for rheumatoid arthritis for which she takes methotrexate, folic acid, ibuprofen and omeprazole.

Which one of the following antibiotics is contraindicated in this patient?

Co-amoxiclav	
Ciprofloxacin	
Cefpodoxime	
Nitrofurantoin	
Trimethoprim	

Submit answer

Reference ranges  $\vee$ 

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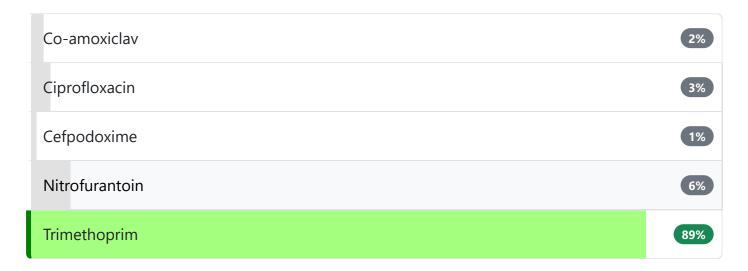


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9. A 39 year old woman presents to her GP with symptoms of dysuria and increased urinary frequency for the past three days. She also complains of lower abdominal pain but has no overt signs of systemic sepsis. Examination is entirely normal aside from mild suprapubic pain. Urine dip correlates with a diagnosis of urinary tract infection with positive nitrites, leukocytes, blood and protein. The sample is sent for culture. The patients medical history is significant only for rheumatoid arthritis for which she takes methotrexate, folic acid, ibuprofen and omeprazole.

Which one of the following antibiotics is contraindicated in this patient?



The concurrent use of methotrexate and trimethoprim containing antibiotics may cause bone marrow suppression and severe or fatal pancytopaenia

Important for me Less important

Trimethoprim is a common bacteriostatic antibiotic used in the treatment of uncomplicated urinary tract infections. It is bacteriostatic rather than bactericidal in that it inhibits bacterial replication rather induces bacterial death. Replication inhibition is achieved by interfering with the generation of thymidine, an essential amino acid base in DNA, by inhibiting the action of the enzyme dihydrofolate reductase which is an essential component of the thymidine metabolic pathway, using folate as a primary substrate. Trimethoprim may also be combined with sulphonamide drugs such as sulphamethoxazole to create antibiotics such as co-trimoxazole; these drugs work synergistically by inhibiting alternative enzymes in the thymidine synthesis pathway.

Although trimethoprim is a relatively safe and effective drug in the treatment of urinary tract infections it does have some contraindications due to its mechanism of action. Due to its antagonism of folate metabolism, it should not be used in pregnancy, particularly the first trimester due to the risk of neural tube defects. Another effect of inhibiting folate metabolism is the impairment of metabolically active tissues such as bone marrow or cancerous cells. This effect is exploited in chemotherapy agents such as methotrexate. This drug also inhibits folate

metabolism to prevent tumour growth; it can also be used in treating rheumatoid arthritis. Concomitant use of methotrexate and trimethoprim can lead to severe and potentially fatal bone marrow suppression with pancytopaenia. This likelihood is increased if the trimethoprim is coupled with a sulphonamide drug, and also in the elderly.

All the other drugs listed above may be used in conjunction with folate inhibiting chemotherapeutic agents, although penicillin containing agents and quinolone antibiotics can cause a reduction in metabolism of methotrexate and the full blood count should be monitored in prolonged courses. Similarly methotrexate and nitrofurantoin used together can cause hepatotoxicity and liver function should be closely monitored. The safest choice of drug above is cefpodoxime, an oral third generation cephalosporin, which has no significant clinical interactions with the other drugs this patient is taking.



Next question >

## Methotrexate \*

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

## Indications

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

## Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - o typically develops within a year of starting treatment, either acutely or subacutely
  - o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

## Prescribing methotrexate

- methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

## Interactions

- avoid prescribing trimethoprim or co-trimoxazole concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

## Methotrexate toxicity

the treatment of choice is folinic acid

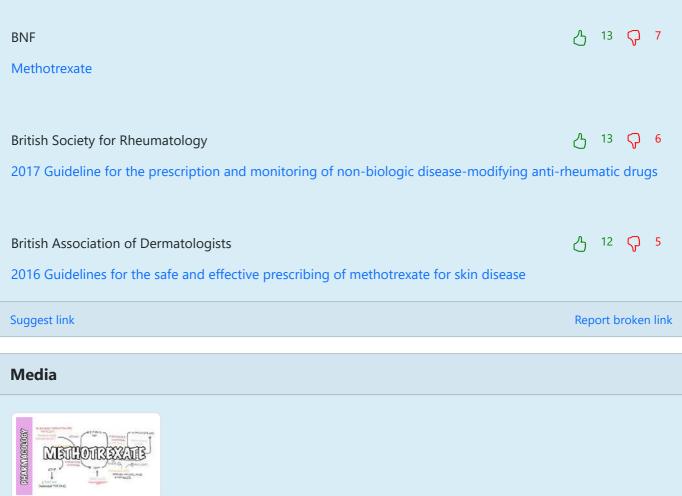


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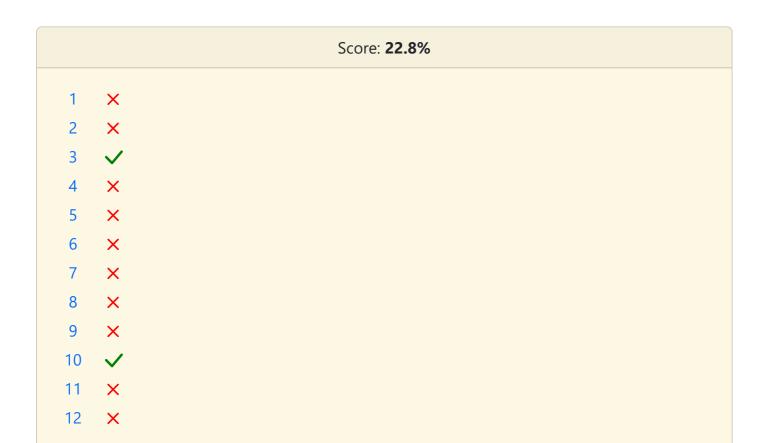




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## Question 73 of 79

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A 40-year-old man is stable on warfarin therapy for the treatment of atrial fibrillation. Whilst on a stag party in Spain he develops scrotal pain and itching. Whilst in Spain, he is treated with a course of ciprofloxacin for a presumed urinary tract infection. Two weeks later he develops a hot, red, swollen and painful knee and both elbows become inflamed. On examination in the emergency department you identify localised tenderness of the knee and painful movement in all directions. The knee is red and hot. Both elbows are mildly warm, with painful movement on flexion and extension. His conjunctiva are also red. He is afebrile. Examination of his external genitalia is essentially normal however there is evidence of excoriation around the scrotum. What is the cause of his knee pain?

Reactive arthritis	
Still's disease	
Septic arthritis	
Gout	
Haemarthrosis	

Submit answer

Reference ranges ∨

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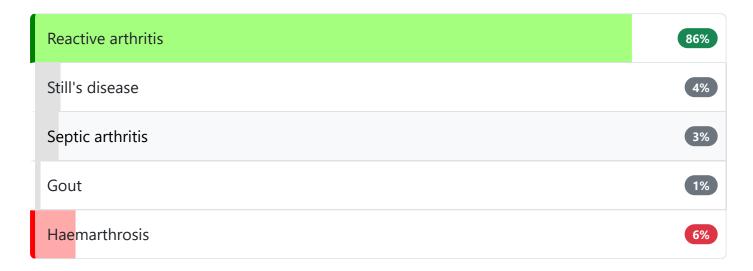








A 40-year-old man is stable on warfarin therapy for the treatment of atrial fibrillation. Whilst on a stag party in Spain he develops scrotal pain and itching. Whilst in Spain, he is treated with a course of ciprofloxacin for a presumed urinary tract infection. Two weeks later he develops a hot, red, swollen and painful knee and both elbows become inflamed. On examination in the emergency department you identify localised tenderness of the knee and painful movement in all directions. The knee is red and hot. Both elbows are mildly warm, with painful movement on flexion and extension. His conjunctiva are also red. He is afebrile. Examination of his external genitalia is essentially normal however there is evidence of excoriation around the scrotum. What is the cause of his knee pain?



Reactive arthritis affects the joints, eyes and genitourinary/gastrointestinal system. It has been associated with gastrointestinal (GI) infections including Shigella, Salmonella, Campylobacter, and other organisms, as well as with genitourinary (GU) infections (especially with Chlamydia trachomatis). The clinical triad commonly occurs 1-4 weeks following exposure.

Ciprofloxacin interacts with warfarin causing prolonged INR which is a risk factor for haemarthrosis, but the history of conjunctivitis and GU symptoms does not support this.



Next question >

## Reactive arthritis: features \*

Reactive arthritis is one of the HLA-B27 associated seronegative spondyloarthropathies. It encompasses what was formerly called Reiter's syndrome\*, a term which described a classic triad of urethritis, conjunctivitis and arthritis following a dysenteric illness during the Second World War. Later studies identified patients who developed symptoms following a sexually transmitted

infection (post-STI, now sometimes referred to as sexually acquired reactive arthritis, SARA).

Reactive arthritis is defined as an arthritis that develops following an infection where the organism cannot be recovered from the joint.

## **Features**

- time course
  - typically develops within 4 weeks of initial infection symptoms generally last around 4-6 months
  - around 25% of patients have recurrent episodes whilst 10% of patients develop chronic disease
- arthritis is typically an asymmetrical oligoarthritis of lower limbs
- dactylitis
- symptoms of urethritis
- eye
  - conjunctivitis (seen in 10-30%)
  - o anterior uveitis
- skin
  - o circinate balanitis (painless vesicles on the coronal margin of the prepuce)
  - keratoderma blenorrhagica (waxy yellow/brown papules on palms and soles)

'Can't see, pee or climb a tree'



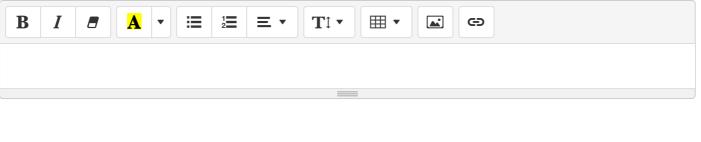
Keratoderma blenorrhagica

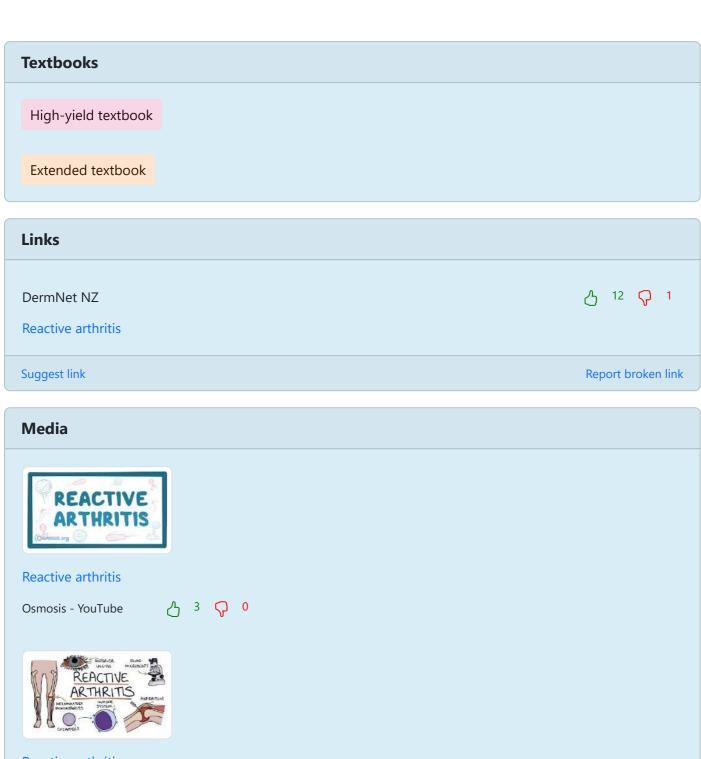
<sup>\*</sup>as Reiter was a member of the Nazi party the term is no longer used



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Reactive arthritis

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Question 74 of 79





A 34-year-old man presents for review. He has been generally unwell since an episode of diarrhoea four weeks ago, with joint pains, pain on passing water and a rash on the soles of his feet:



What does this rash likely represent?

Pompholyx	
HIV-associated dermopathy	
Plantar pustular psoriasis	
Mosaic warts	
Keratoderma blennorrhagica	

Submit answer

Reference ranges ✓

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Question 74 of 79



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A 34-year-old man presents for review. He has been generally unwell since an episode of diarrhoea four weeks ago, with joint pains, pain on passing water and a rash on the soles of his feet:



What does this rash likely represent?



The correct answer is **keratoderma blennorrhagica**. Keratoderma blennorrhagica is a skin condition associated with reactive arthritis, which is an autoimmune response to an infection in another part of the body. The patient's history of diarrhoea, joint pains, and pain on passing water suggests that he may have reactive arthritis. The rash on the soles of his feet supports this diagnosis as keratoderma blennorrhagica typically presents as brownish-red hyperkeratotic plaques on the soles and occasionally palms.

**Pompholyx** is an incorrect answer because it presents as small, intensely itchy vesicles on the fingers, palms, and soles. It does not match the description or appearance of the rash seen in this case.

HIV-associated dermopathy is also incorrect because it usually presents as a widespread pruritic

rash involving multiple body sites rather than being confined to the soles. Moreover, there are no other clinical features mentioned in the case that would suggest HIV infection.

**Plantar pustular psoriasis** can be ruled out because although it affects the soles of feet and can cause discomfort during walking, it typically presents with pustules and scale formation which are not observed in this case.

Finally, **mosaic warts** are an incorrect answer because they present as grouped verrucae (warts) with a rough surface on weight-bearing areas of the foot. These warts are caused by human papillomavirus (HPV) infection and do not fit with the clinical picture described for this patient.



Next question >

# Reactive arthritis: features \*

Reactive arthritis is one of the HLA-B27 associated seronegative spondyloarthropathies. It encompasses what was formerly called Reiter's syndrome\*, a term which described a classic triad of urethritis, conjunctivitis and arthritis following a dysenteric illness during the Second World War. Later studies identified patients who developed symptoms following a sexually transmitted infection (post-STI, now sometimes referred to as sexually acquired reactive arthritis, SARA).

Reactive arthritis is defined as an arthritis that develops following an infection where the organism cannot be recovered from the joint.

## **Features**

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  - conjunctivitis (seen in 10-30%)
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  - keratoderma blenorrhagica (waxy yellow/brown papules on palms and soles)

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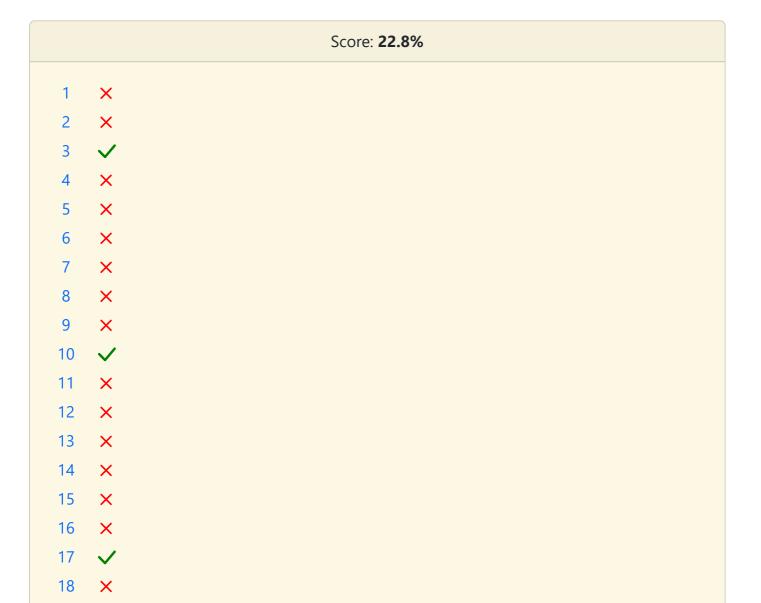
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Question 75 of 79

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A 52-year-old woman presents with several months' history of fatigue and dry eyes. She also describes a significantly dry mouth, to the point that she sometimes finds it difficult to eat food. She has no significant past medical history.

On examination, the eyes appear mildly red. The tongue appears dry. Given the suspected diagnosis, the doctor requests an initial blood test:

Antinuclear antibodies 1:1600 (<1:280)

What is the most definitive test to confirm the likely diagnosis?

Anti-La antibody titre	
Anti-Ro antibody titre	
Salivary gland biopsy	
Schirmer's test	
Sialometry	

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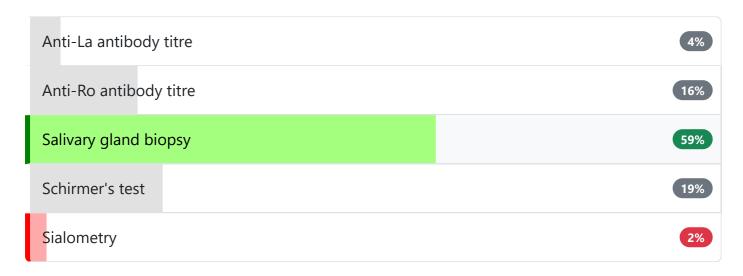


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On examination, the eyes appear mildly red. The tongue appears dry. Given the suspected diagnosis, the doctor requests an initial blood test:

Antinuclear antibodies 1:1600 (<1:280)

What is the most definitive test to confirm the likely diagnosis?



Salivary gland biopsy is the most definitive way of confirming the diagnosis of primary Sjogren's syndrome - sections will show a typical lymphocytic infiltrate

Important for me Less important



The diagnosis here is that of Sjogren's syndrome, an autoimmune disorder affecting exocrine glands and resulting in dry mucosal surfaces. The high antinuclear antibody titre is non-specific, but reinforces the diagnosis of an autoimmune condition and is present in 70% of patients. Of the options listed above, the most definitive diagnostic tool is salivary gland biopsy. This will show a focal lymphocytic infiltration.

Anti-La antibodies are found in 30% of patients with primary Sjogren's syndrome. This is not particularly specific or sensitive for the condition, and certainly not as definitive a test as biopsy.

Similarly, **anti-Ro antibodies** are not entirely specific or sensitive for Sjogren's syndrome. They are found in 70% of patients, rather than the 30% seen with anti-La, but are still not as definitive as biopsy.

**Schirmer's test** is a quantitative measurement of tears using filter paper near the conjunctival sac. It is useful to aid diagnosis but is not as definitive as salivary gland biopsy.

**Sialometry** measures unstimulated salivary flow for 15 minutes into a calibrated tube. This is useful to aid diagnosis but does not differentiate between causes of dry mouth, of which Sjogren's syndrome is just one cause.



Next question >

# Sjogren's syndrome 🖈

Sjogren's syndrome is an autoimmune disorder affecting exocrine glands resulting in dry mucosal surfaces. It may be primary (PSS) or secondary to rheumatoid arthritis or other connective tissue disorders, where it usually develops around 10 years after the initial onset. Sjogren's syndrome is much more common in females (ratio 9:1). There is a marked increased risk of lymphoid malignancy (40-60 fold).

### **Features**

- dry eyes: keratoconjunctivitis sicca
- dry mouth
- vaginal dryness
- arthralgia
- Raynaud's, myalgia
- sensory polyneuropathy
- recurrent episodes of parotitis
- renal tubular acidosis (usually subclinical)

# Investigation

- rheumatoid factor (RF) positive in nearly 50% of patients
- ANA positive in 70%
- anti-Ro (SSA) antibodies in 70% of patients with PSS
- anti-La (SSB) antibodies in 30% of patients with PSS
- Schirmer's test: filter paper near conjunctival sac to measure tear formation
- histology: focal lymphocytic infiltration
- also: hypergammaglobulinaemia, low C4

# Management

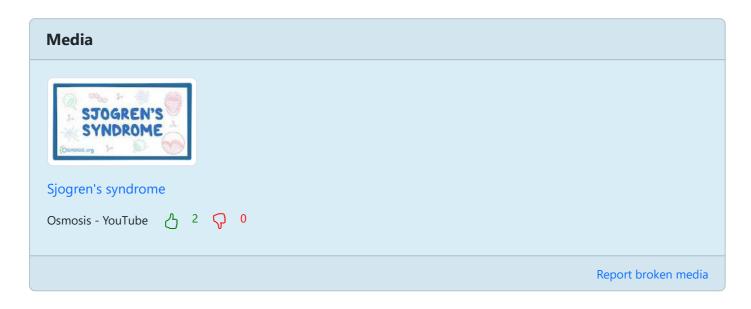
- artificial saliva and tears
- pilocarpine may be helpful to stimulate saliva production

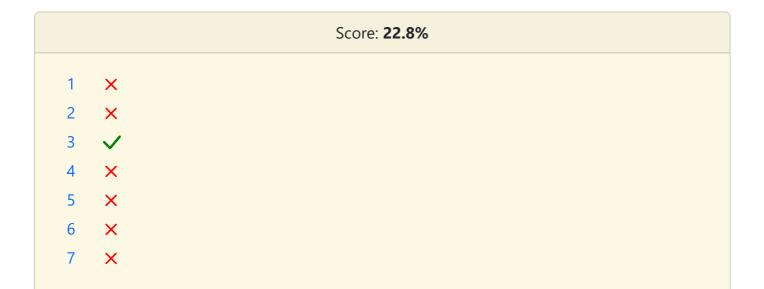


Next question >









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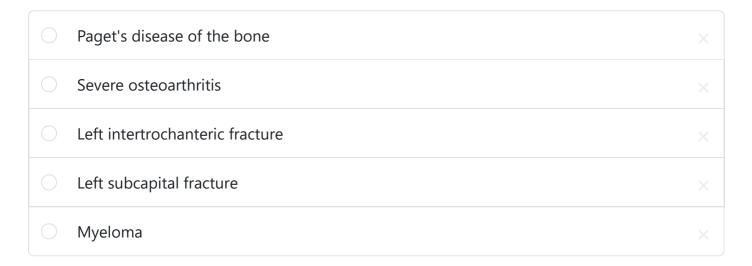




An 80-year-old man presents is taken to the Emergency Department after falling at home. His daughter notes that he fell onto his left side. An x-ray is taken of the pelvis:



# What is the diagnosis?



Submit answer

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Question 76 of 79



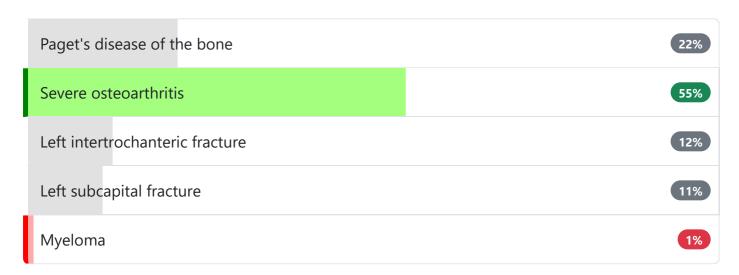
 $\square$ 



An 80-year-old man presents is taken to the Emergency Department after falling at home. His daughter notes that he fell onto his left side. An x-ray is taken of the pelvis:



# What is the diagnosis?



This x-ray shows advanced osteoarthritic changes at the left hip joint; loss of joint space and subchondral sclerosis are prominent.



# Osteoarthritis: x-ray changes \*

X-ray changes of osteoarthritis (LOSS)

- Loss of joint space
- Osteophytes forming at joint margins
- **S**ubchondral sclerosis
- **S**ubchondral cysts



Next question >







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Back to top







Question 77 of 79



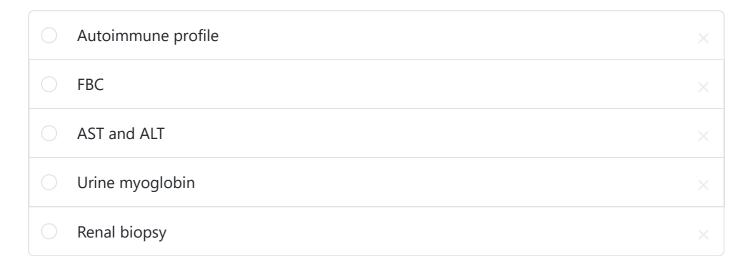


An 80-year-old retired GP with no past medical history presents to the hospital with a 6-month history of muscle aches and weakness. She also has difficulty swallowing and has had 3 courses of antibiotics for a presumed chest infection in the last 3 months. In the last 2 days she has been struggling to cope at home and has had two falls.

Blood tests show:

Erythrocyte Sedimentation Rate (ESR) 60 mm/hour g/l
Creatinine Kinase 8000 U/L

Which of the following investigations would be LEAST helpful in the workup?



Submit answer

Reference ranges ∨

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Question 77 of 79



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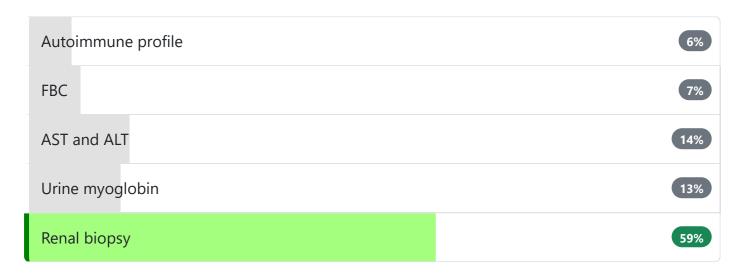


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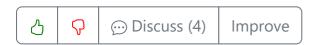
### Blood tests show:

Erythrocyte Sedimentation Rate (ESR) 60 mm/hour g/l Creatinine Kinase 8000 U/L

Which of the following investigations would be LEAST helpful in the workup?



This lady seems to have polymyositis. An autoimmune profile is useful as ANA is positive in one-third of patients. Anti-jo antibodies are positive in 20% of patients and indicate a poorer prognosis with interstitial lung disease. A full blood count may show leukocytosis or thrombocytosis. AST/ALT are both muscle enzymes that will be elevated. Creatinine kinase will also be elevated along with urine myoglobin. In some cases, the patient may develop renal impairment from rhabdomyolysis and thus U+Es should be monitored. A renal biopsy would not be diagnostically helpful in this situation.



Next question >

### Overview

- inflammatory disorder causing symmetrical, proximal muscle weakness
- thought to be a T-cell mediated cytotoxic process directed against muscle fibres
- may be idiopathic or associated with connective tissue disorders
- associated with malignancy
- dermatomyositis is a variant of the disease where skin manifestations are prominent, for example a purple (heliotrope) rash on the cheeks and eyelids
- typically affects middle-aged, female:male 3:1

## **Features**

- proximal muscle weakness +/- tenderness
- Raynaud's
- respiratory muscle weakness
- interstitial lung disease
  - o e.g. fibrosing alveolitis or organising pneumonia
  - o seen in around 20% of patients and indicates a poor prognosis
- dysphagia, dysphonia

# Investigations

- elevated creatine kinase
- other muscle enzymes (lactate dehydrogenase (LDH), aldolase, AST and ALT) are also elevated in 85-95% of patients
- EMG
- muscle biopsy
- anti-synthetase antibodies
  - anti-Jo-1 antibodies are seen in pattern of disease associated with lung involvement,
     Raynaud's and fever

# Management

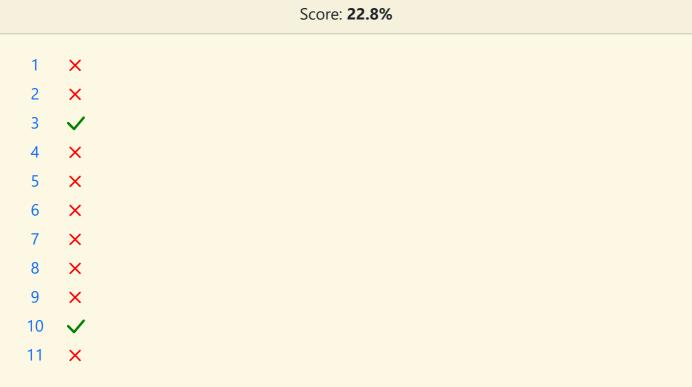
- high-dose corticosteroids tapered as symptoms improve
- azathioprine may be used as a steroid-sparing agent



Next question >



# **Textbooks** High-yield textbook Extended textbook Media Polymyositis and Dermatomyositis in 3 Minutes Townsend Teaching - YouTube Understanding Myositis (Polymyositis and Dermatomyositis) 台 ○ ♀ 1 Zero To Finals - YouTube Report broken media Score: 22.8% X



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A 56-year-old man is seen in the emergency department with a 2-week history of worsening fatigue and exertional shortness of breath. He denies any recent infective symptoms or syncopal episodes. His past medical history includes Crohn's disease, for which he was recently started on azathioprine, type 2 diabetes, epilepsy, and gout.

On examination, he is pale. Chest sounds are clear and heart sounds are normal. He has a soft and non-tender abdomen.

# Laboratory tests:

Hb	81 g/L	(135 - 180)
Platelets	66 * 10 <sup>9</sup> /L	(150 - 400)
WBC	1.1 * 10 <sup>9</sup> /L	(4.0 - 11.0)
Neuts	0.3 * 10 <sup>9</sup> /L	(2.0 - 7.0)
Na <sup>+</sup>	136 mmol/L	(135 - 145)
K <sup>+</sup>	4.1 mmol/L	(3.5 - 5.0)
Urea	3.1 mmol/L	(2.0 - 7.0)
Creatinine	83 µmol/L	(55 - 120)

Given this patient's presentation, what medication should be stopped?

	Allopurinol	
	Gliclazide	×
0	Lansoprazole	×
0	Metformin	×
	Sodium valproate	

Submit answer

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Question 78 of 79



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On examination, he is pale. Chest sounds are clear and heart sounds are normal. He has a soft and non-tender abdomen.

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Hb	81 g/L	(135 - 180)
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K <sup>+</sup>	4.1 mmol/L	(3.5 - 5.0)
Urea	3.1 mmol/L	(2.0 - 7.0)
Creatinine	83 µmol/L	(55 - 120)

Given this patient's presentation, what medication should be stopped?

Allopurinol	71%
Gliclazide	2%
Lansoprazole	2%
Metformin	3%
Sodium valproate	22%

Azathioprine and allopurinol have a severe interaction causing bone marrow suppression

| Important for me | Less important |

This patient's blood results show pancytopenia with low haemoglobin, platelets and white cell count. He is also experiencing symptoms of anaemia with increasing lethargy and exertional shortness of breath. When investigating a patient with symptomatic pancytopenia, it is important

to exclude any iatrogenic causes, such as medication. This patient has recently been started on azathioprine, an immunosuppressant medication. Of the drugs listed in this question, azathioprine must not be given in conjunction with **allopurinol** as the 2 drugs cause an increased risk in haematological toxicity, such as that seen in this patient.

**Gliclazide** is a sulfonylurea used in the treatment of diabetes mellitus. The most important side effect to note is the risk of hypoglycaemia.

**Lansoprazole** is a proton pump inhibitor used in the treatment of gastro-oesophageal reflux disease. Side effects include electrolyte imbalances such as hypomagnesaemia and an increase in the risk of *Clostridium difficile* infection.

**Metformin** is used in the treatment of diabetes mellitus. Gastrointestinal side effects are the most common including diarrhoea and abdominal pain. It does not interact with azathioprine.

**Sodium valproate** is an anti-epileptic medication that has a number of side effects including pancreatitis, weight gain, alopecia and hepatic toxicity. Rarely, it can cause agranulocytosis. However, it does not interact with azathioprine and would not be contraindicated in this patient.



Next question >

# Azathioprine \*

Azathioprine is metabolised to the active compound mercaptopurine, a purine analogue that inhibits purine synthesis. A thiopurine methyltransferase (TPMT) test may be needed to look for individuals prone to azathioprine toxicity.

Adverse effects include

- bone marrow depression
  - consider a full blood count if infection/bleeding occurs
- nausea/vomiting
- pancreatitis
- increased risk of non-melanoma skin cancer

A significant interaction may occur with allopurinol and hence lower doses of azathioprine should be used.

Azathioprine is generally considered safe to use in pregnancy.





## **Textbooks**

High-yield textbook

Extended textbook

## Media



Azathioprine - Pharmacology, mechanism of action, side effects

Armando Hasudungan - YouTube

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Score: 22.8%

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## Question 79 of 79





A 65-year-old lady is admitted urgently to hospital after her General Practitioner noted an abnormality in routine bloods. She is known to suffer from rheumatoid arthritis. Current medications include methotrexate 15 mg once per week, folic acid 5 mg (6 days of the week), ramipril 2.5 mg OD, atorvastatin 40 mg OD. She was recently commenced on antibiotics for a urinary tract infection. The bloods are the following:

Hb	68 g/l
Platelets	65 * 10 <sup>9</sup> /I
WBC	2.1 * 10 <sup>9</sup> /l
Neutrophils	0.5 * 10 <sup>9</sup> /l
Lymphocytes	0.7 * 10 <sup>9</sup> l
Monocytes	0.1 * 10 <sup>9</sup> l

Which antibiotic prescribed for this lady could explain this picture?

Nitrofurantoin	
Trimethoprim	
Co-amoxiclav	
Cefalexin	
Ciclosporin	

Submit answer

Reference ranges ∨

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Question 79 of 79



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A 65-year-old lady is admitted urgently to hospital after her General Practitioner noted an abnormality in routine bloods. She is known to suffer from rheumatoid arthritis. Current medications include methotrexate 15 mg once per week, folic acid 5 mg (6 days of the week), ramipril 2.5 mg OD, atorvastatin 40 mg OD. She was recently commenced on antibiotics for a urinary tract infection. The bloods are the following:

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Lymphocytes	0.7 * 10 <sup>9</sup> l
Monocytes	0.1 * 10 <sup>9</sup> l

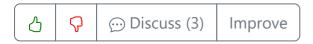
Which antibiotic prescribed for this lady could explain this picture?



The concurrent use of methotrexate and trimethoprim containing antibiotics may cause bone marrow suppression and severe or fatal pancytopaenia

Important for me Less important

Methotrexate is a folic acid antagonist affecting the cell cycle. Trimethoprim is also a folic acid antagonist. When combined together the risk of myelosuppression is increased, given also the fact that trimethoprim reduces the renal excretion rate of methotrexate.



## Methotrexate \*

Methotrexate is an antimetabolite that inhibits dihydrofolate reductase, an enzyme essential for the synthesis of purines and pyrimidines. It is considered an 'important' drug as whilst it can be very effective in controlling disease the side-effects may be potentially life-threatening - careful prescribing and close monitoring is essential.

#### Indications

- inflammatory arthritis, especially rheumatoid arthritis
- psoriasis
- some chemotherapy acute lymphoblastic leukaemia

#### Adverse effects

- mucositis
- myelosuppression
- pneumonitis
  - the most common pulmonary manifestation
  - similar disease pattern to hypersensitivity pneumonitis secondary to inhaled organic antigens
  - o typically develops within a year of starting treatment, either acutely or subacutely
  - o presents with non-productive cough, dyspnoea, malaise, fever
- pulmonary fibrosis
- liver fibrosis

#### Pregnancy

- women should avoid pregnancy for at least 6 months after treatment has stopped
- the BNF also advises that men using methotrexate need to use effective contraception for at least 6 months after treatment

#### Prescribing methotrexate

- methotrexate is a drug with a high potential for patient harm. It is therefore important that you are familiar with guidelines relating to its use
- methotrexate is taken weekly, rather than daily
- FBC, U&E and LFTs need to be regularly monitored. The Committee on Safety of Medicines recommend 'FBC and renal and LFTs before starting treatment and repeated weekly until therapy stabilised, thereafter patients should be monitored every 2-3 months'
- folic acid 5mg once weekly should be co-prescribed, taken more than 24 hours after methotrexate dose
- the starting dose of methotrexate is 7.5 mg weekly (source: BNF)
- only one strength of methotrexate tablet should be prescribed (usually 2.5 mg)

- avoid prescribing trimethoprim or co-trimoxazole concurrently increases risk of marrow aplasia
- high-dose aspirin increases the risk of methotrexate toxicity secondary to reduced excretion

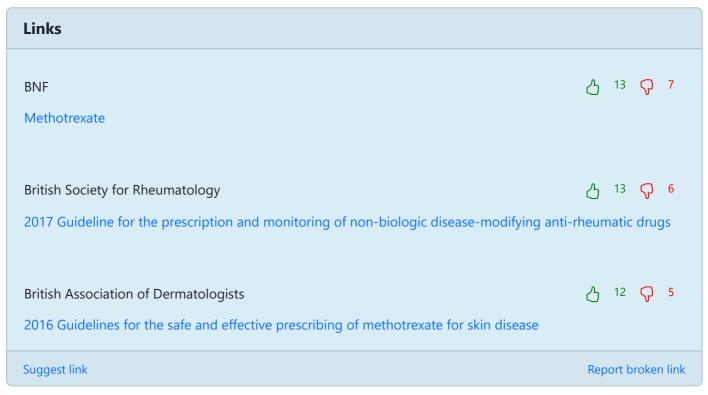
## Methotrexate toxicity

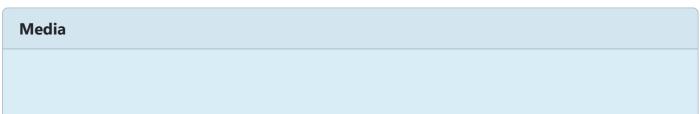
• the treatment of choice is folinic acid













Methotrexate - Pharmacology (DMARDs, mechanism of action, side effects)

Armando Hasudungan - YouTube





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